PULMONARY HÆMORRHAGE

THOMPSON

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THE CAUSES AND RESULTS

OF

PULMONARY HÆMORRHAGE

WITH REMARKS ON TREATMENT

BY

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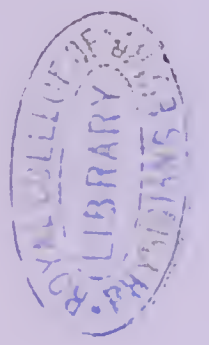

With Illustrations

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PREFACE.

He who peruses these pages may perhaps find something written in them which is not in accordance with the accepted doctrines of the great masters of Medicine, and so he may deem my remarks unworthy of his fair consideration.

I entreat him not to think that the opinions here advanced are put forward with haste, or in a careless spirit: they are the results of some industrious diligence extending over many years devoted especially to the investigation of the subject; and what is here set forth is the outcome of the physical examination of over twenty-two thousand patients during their lifetime, and of the inspection of three hundred cases of diseases of the chest after the death of the patients.

Let me ask him then to read these pages without disdain, and if he takes exception to the conclusions, and proves my errors—which must, I know, be manifold—I will gladly make all the amends in my power towards the knowledge of the truth and the advancement of medical science.

January 1879.

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PULMONARY HÆMORRHAGE.

CHAPTER I.

RETROSPECTIVE.

Few convictions on medical subjects are more deeply impressed on the minds even of the uneducated than that the connection between consumption and blood-spitting is so inseparable, that when either is developed, the other cannot be long delayed, and is sure eventually to be established, closely treading in the footsteps of its constant companion.

But when the subject is considered from the scientific aspect, and the history of past and present opinions is investigated, it will be found that diverse and even opposite doctrines have been advanced, not as to the frequent association of blood-spitting with consumption, for on this point there is indeed no room for a difference of opinion, but on the exact relation which each bears to the other as to cause and effect.

Yet there are not many points of medical evidence which can be accepted as so trustworthy as the evidence with regard to blood-spitting: the unwonted sight of blood, the certainty of the event, the manifest danger to the patient while the life blood is flowing—all combine to produce a vivid effect upon the observer not likely soon to be effaced, and it seems strange that in spite of the large number of cases which must have afforded ample opportunities for observation, there should be at the present time any difference of opinion on the subject; but in truth it is this very frequent association of the two conditions which presents the great obstacle in arriving at a decisive

conclusion, the difficulty being to find a sufficient number of cases in which pulmonary hæmorrhage can be studied separately from a condition of phthisis. A retrospective glance at the different doctrines which have been maintained, from the earliest days of medicine to the present time, will show the diversity of opinion with which the subject has been treated, and the different views will be found to fall readily into certain groups, according to the arguments used.

The earliest opinion on the subject is to be found in the aphorisms of Hippocrates, whose authority not only represents the doctrines prevalent during his own time, but moulded those of many subsequent ages: this opinion was founded upon a simple argument that, as pus so generally follows blood, what more probable than that blood remaining in the lungs after an attack of blood-spitting should become pus, and thus induce the pulmonary disease phthisis?

Centuries after this doctrine had been put before the world of science, the subject was more fully considered in England by Richard Morton, who in his 'Phthisiologia,' published in 1727, devoted more than one chapter to the consideration of *Phthisis ab Hæmoptoe.*

He commences his chapter on the subject by the remark that the well-known adage that pus follows blood is probably due, before all things, to the rapidity with which phthisis follows hæmoptysis, and although he expresses no decided opinion as to the process by which phthisis is established, he records the theories which had passed through his mind as possible, in the following passage:—

'Quod utrum a crasi sanguinis ob nimium ejus dispendium eversâ vel a copioso humorum ex universo corporis habitu, quâ datur via in teneros pulmones affluxu: vel a sanguinis grumosi in ipsis pulmonibus post hæmoptoen relicta putrefactione: vel tandem ab ulcere aliquo erosionem vasorum insequenti accidat, parum scio.'

The question evidently appears to him involved and intricate, while considerable thoughtfulness is shown in the theories advanced. The cases given in illustration are well chosen and striking, and the pathological appearances recorded are curiously exact and accurate, so that, although they receive no interpreta-

tion at the hands of Morton, they afford remarkable proofs of his powers of observation.

But these opinions held during the last century gave way before the subsequent teachings of Laennec, who has influenced in an extraordinary manner the writers of the present century, not only in this special point, but in others connected with phthisis.

There is no evidence in the works of Laennec that all the complicated conditions which involve the subject had been fully considered, and the possibility of phthisis being a result of hæmoptysis is negatived on evidence which is unsatisfactory, because it is negative in kind. But the arguments adduced are, as Niemeyer justly points out, fallacious. Blinded by the idea that tubercle is in all cases the primary stage of phthisis, Laennec confined his observations to the production of tubercle by blood, and he was ultimately driven to a negative conclusion.

Louis took the same view as Laennec, and quotes Fournet as adopting what he terms the obsolete notion that hæmoptysis was an exciting cause of phthisis.

Andral, on the other hand, finds more difficulty in answering the question, and gives details of a case which, he considered, pointed to a different conclusion.

English writers of the present century have generally accepted the teachings of Laennec, and have put aside, after a very incomplete consideration of the subject, the theory involved in a *phthisis ab hæmoptoe*. I am speaking now of those authors who have in great measure tended to form the opinions of the present generation, and who are regarded as authoritative on the subject. The only man, as far as I am aware, who stands out as a brilliant exception, is Graves, of Dublin, who, in his Clinical Lectures, devotes a chapter to hæmoptysis. His views are original and remarkable. He thought that blood effused into the lung tissue might remain without producing any particular symptom ; that where scrofula existed, tubercles (to quote his own words) might be precipitated into suppuration, and he gives two cases of severe hæmoptysis which he had carefully watched and in which a condition of suppurating pneumonia was found after death.

To the question, Can spitting of blood be considered as the

cause of consumption? he gives the following answer:—'In more than one case of pulmonary apoplexy in which the patient died of the first attack not a single tubercle could be found in the lungs. It may certainly produce a tendency to consumption, but it is not a necessary cause of it, and the same thing may be said of bronchial hæmoptysis.'

He differs from those who, like Morton, think that effused blood may become corrupted and a cause of gangrene.

These views of Graves are important and are based upon careful pathological observations: it is strange that they have not attracted more attention, as they are a nearer approach to a true solution of the problem than any theories hitherto broached by other observers.

In Germany the subject has been taken up on the lines of the theory propounded by Morton and Hoffmann, the now well-known views of Niemeyer having recently revived attention to the question: to quote Niemeyer's words, bronchial bleeding may precede the development of consumption as its cause, the hæmorrhage leading to chronic inflammation and destruction of the lung.

The cases which are brought forward in illustration and proof of this notion are important, the examination of the peculiar conditions reported leading to the conclusion that caseous pneumonia resulted from the presence of blood in the air cells.

This view has been supported by Weber and Bäumler, in England, in two papers published in the Transactions of the Clinical Society. It would appear, however, that this theory has not been accepted generally in Germany, and has indeed been controverted by experiments on animals which have resulted in negative conclusions; and if the opinions expressed in Ziemssen's 'Cyclopædia' may be taken to represent in any measure the present state of opinion in Germany, the conclusion of Hertz, who says 'we must deny that hæmorrhage produces phthisis,' lands us exactly in the same place in which we are left by the opinions of Laennec.

If we look across the Atlantic, we shall find that the same answer to the question is given by Flint of New York, in his recent work on phthisis: so that, turn in what direction we may,

it is evident that the doctrines of Laennec still maintain their sway.

Negative conclusions must however give way to positive facts, and those who can find no effects resulting from hæmoptysis must explain away, in some manner, the very remarkable pathological conditions which are described with such exactness in the works of Morton, Graves, Niemeyer, and others; and it is from pathology that the problem must receive its solution, for from a clinical point of view the difficulties are so great that some sure evidence must be first obtained from morbid anatomy, before we are in a position to decide what we ought to look for: yet the issues are so important that the problem must be solved, and an answer be found, for that question which is so often asked of the physician, What is the relation of blood-spitting to consumption?

I shall endeavour to unravel some of the obscurity which envelopes the subject, in that order of events in which it has been presented to my notice.

For some years I devoted special attention to the elucidation of the subject from a clinical point of view, but I failed at arriving at a satisfactory solution, until a clue was first obtained from pathology. The pathological results of bleeding from the lungs being once established, it became an easier task to follow the significance of clinical signs and symptoms; and so to take the subject in order, it seems best to consider—

1. The sources of pulmonary bleedings.
2. The pathology of the subject.
3. Its clinical aspects.

I would here express my thanks to my friends and colleagues for the kindness and courtesy with which they have placed their cases at my disposal; without this freedom, which they have so willingly accorded to me, cases which have formed intermediate links might have been lost to me, and the thread of connection between various points in the subject might have been broken, and it is only by the repeated examination of cases in all stages of disease that any decision could be made in what is, no doubt, an extremely complicated subject.

In all discussions respecting phthisis and tubercle it is necessary to define precisely the meaning attached to the terms

used: and such a definition is the more necessary in this place, as my views of the correlation of phthisis and tubercle differ from those which have been, and are now, generally maintained.

Phthisis appears to me to be that form of destructive disease which terminates a variety of pulmonary diseases which have their origin in inflammation and irritation: it is the common result to which these diseases tend to converge, which they may ultimately assume, and in which they lose their initial peculiarities.

With regard to the use of the word 'tubercle,' it is used here in a limited sense—the nodular form known to Laennec as grey tubercle, which appears to me to be the result of secondary infection, whether it be considered as a general infection of the lungs, as it occurs in acute tuberculosis, or as a more local infection restricted to portions of the lungs. The latter form is that in which it is generally presented to view in the post-mortem examinations of phthisical lungs, and I believe it results in many cases from infection with septic matter, which is derived from a secreting cavity, drawn over the bronchial tubes into the lobules by the force of insufflation, of which force as an agent, in transplanting matter from one place to another in the lungs, some proof will be given in the following pages.

It will not be supposed that I mean by this to convey the idea that the bronchial tubes are the only carriers of infective matter, but it would seem that their influence has been overlooked.

With the diffused tubercular condition as an early condition of some forms of phthisis, the present arguments are not exceedingly concerned, but this appears to me to be caused by septic alterations of inflammatory products *in situ*, due in great probability to the non-removal of such products.

It is impossible for me here, as it would be altogether out of place, to enter into the various arguments and evidence that have decided me in forming these conclusions—evidence and arguments will be brought forward in these pages which are germane to the subject, and may perhaps prove some justification for the views just expressed regarding the correlation of phthisis and tubercle.

CHAPTER II.

THE SOURCES OF PULMONARY HÆMORRHAGE.

The first point to which attention must be directed is the arrangement and peculiar distribution of blood vessels in the lungs, which are known to all anatomists, but demand some consideration before any attempt can be made to discuss the various sources from which pulmonary hæmorrhage is likely to flow. The following short description, which is based upon the accounts of others (more especially upon those of Professor Turner, in his 'Introduction to Anatomy'), will serve the purpose I have in view.

The function of the lungs and their nutrition render a double circulation of blood necessary: the functional supply, which is far the larger of the two, being obtained through the pulmonary artery, the nutritive supply being furnished by the bronchial arteries.

The pulmonary artery, which springs directly from the right ventricle of the heart, enters the lungs between the bronchial tube and the pulmonary vein of either side. The artery then passing with the bronchial tubes to the lobules, subdivides rapidly, without forming any anastomoses with its fellows, and terminates in the plexus of capillaries which surround the air cells. These capillaries are very fine and close, and extremely superficial, being covered only by tesselated epithelium.

The pulmonary veins arise chiefly from these capillaries, and after running together to form larger branches, finally emerge from each lung by two large veins, which empty themselves into the left auricle.

These veins have no valves and freely anastomose.

The bronchial arteries, from one to three for each lung, spring from the aorta or from an intercostal branch, and are

distributed to the bronchial glands, the coats of the large blood-vessels, the fibrinous and muscular coats of the bronchial tubes, the interlobular connective tissue, finally forming a delicate capillary plexus which supplies the mucous membrane of the bronchial tubes—the branches which supply the smallest bronchioles communicating with the pulmonary capillary plexus.

The blood supplied by the bronchial arteries is returned partly by the bronchial veins, which are not so capacious as the arteries, and partly by the pulmonary veins. The bronchial veins unite at the root of the lung, and on the right side pass into the vena azygos, and in the left into the superior intercostal vein.

Artificial injections can readily be forced from the bronchial arteries into the pulmonary capillaries, but the bronchial arteries cannot be injected from the pulmonary artery: as an additional proof of the inter-connection of the bronchial arteries and pulmonary capillaries, it may be well to mention that a case is recorded by Virchow, in which the bronchial system was found enlarged consequent upon an impediment in the pulmonary supply.

The dark venous blood, which is supplied by the pulmonary artery, becomes readily aërated by exposure in the capillaries to the inspired air; the blood supplied by the bronchial vessels being red in colour, is identical with that in the pulmonary capillaries and pulmonary veins: and inasmuch as bronchial capillaries are freely exposed to air, and a great portion of the blood is transferred into the pulmonary veins, which carry arterial blood, it is at least possible that the blood of the bronchial veins is not very different in colour.

I am not aware, however, of any experiments which bear upon this point; but I may remark that the venous hue, which is seen in bronchial congestion, is very different from the delicate pink which would appear to be the colour of the tubes in a state of simple irritation. What I wish to point out is this, that the pulmonary capillaries and pulmonary veins circulate arterial blood, as well as the bronchial arteries and bronchial capillaries, under conditions of health, so that he must indeed be a bold man who ventures to decide upon the source of a

hæmorrhage from a consideration solely of the colour of the blood ejected.

But this difficulty is greatly enhanced by the alteration which the blood undergoes through conditions of disease, and while the ejection of blood may on the one hand change pulmonary venous blood to arterial, on the other hand arterial blood may be darkened by its detention in the lungs. Attention must also be called to the facts that pulmonary veins contain a far larger supply of blood than the bronchial arteries, and not only are the pulmonary plexus the finest and closest in the body, but they are also more exposed and less supported than the bronchial capillaries.

The terms bronchorrhagia and pneumorrhagia have been suggested to indicate the special origin of the blood in different cases; but this suggestion is not a wise one, as it is impossible in many cases of trivial hæmorrhage to decide from which set of vessels the blood has flowed.

But this terminology is also objectionable, because it indicates a further development of the ideas of Laennec, who used the term bronchial hæmorrhage to indicate that form 'qui dépend d'un simple suintement du sang à la surface interne des bronches.' In the passage in his work which follows this sentence, he goes on to say that the ancients attributed hæmoptysis to the rupture of the vessels of the lungs, but that this theory adopted without sufficient proof has been too absolutely rejected by educated physicians of the day: and he continues in the following remarkable passage, which shall be given in his own words: 'Il n'est point impossible qu'un anévrisme d'un des rameaux d'artère pulmonaire ou des veines se développent et donnent lieu à une hémorrhagie, quoiqu'il n'existe, au moins à ma connaissance, aucun fait bien décrit de ce genre.'

He concludes that in the actual condition of science, at that time, it could be affirmed that slight hæmoptysis took place by a simple diapedesis or exhalation of blood at the surface of the bronchial membrane, and those of a grave nature had their origin in the vesicular tissue of the lungs, and constitue that affection to which he gave the name of pulmonary apoplexy.

It is difficult to understand how Laennec could have overlooked the following exceptions to his conclusions: hæmorrhage

slight in quantity arises from the pulmonary capillaries in pneumonia, in brown induration of the lung in congestion, and even in some cases of pulmonary apoplexy, while profuse hæmorrhage may take place from isolated vessels, as in some aneurisms of the aorta, as well as in erosion of a large vessel from the destructive action of phthisis.

The pathological grounds on which the theory of hæmorrhage proceeding from the bronchial tubes is based are decidedly fallacious.

Laennec says that, when bodies are examined of those who have succumbed to a bronchial hæmorrhage, or at the moment when they are attacked by hæmorrhage, a greater or less quantity of blood, liquid or coagulated, is found in the bronchial tubes: the bronchial mucous membrane is impregnated with blood and stained, and generally slightly softened.

Now this is without doubt a very accurate description of the condition of the bronchial tubes in cases of fatal hæmoptysis, but the deduction that the blood has proceeded from the bronchial mucous membrane can be easily shown to be erroneous. This condition is simply due to the regurgitation of blood into the lungs at the moment of death, whatever be the source from which the blood has come.

For example, I have examined many cases of fatal hæmorrhage in which the blood has been traced to a pulmonary vessel eroded or in a state of aneurism, to the bursting of an aortic aneurism into the trachea, to aneurism of the coronary artery of the stomach: in all these cases blood was found in the bronchi; moreover, it is difficult to determine, by inspection of the condition of the bronchial tubes, from which lung the blood has started in cases of pulmonary hæmorrhage, although the actual source may be subsequently determined: it can thus be positively asserted that the presence of blood in the bronchial tubes does not establish the existence of bronchial hæmorrhage; it shows in a remarkable manner the tendency of the inspiratory force to translate matter from one lung to the other, or from one part to another—a point which will receive further illustration when the pathological evidence of hæmorrhage is subsequently considered.

In no case of profuse hæmorrhage have I been able to satisfy myself that the blood proceeded solely from the bronchial

vessels: one such case has been put upon record, but in this the blood proceeded from an aneurism of the bronchial artery—a very rare condition; in another case of profuse hæmorrhage that I examined, some of the blood proceeded from the bronchial mucous membrane, but also from the pulmonary vessels, due to a general tendency to hæmorrhage. The proof of bronchial hæmorrhage depends upon the condition of the vessels, which are enlarged and well marked in undoubted instances of the kind; and this evidence is only present, as far as I am aware, in cases of bronchitis and bronchial congestion; in these cases of irritation which arise from the presence of septic matter derived from neighbouring ulceration of the lung, and in cases of general pulmonary hæmorrhage.

Putting aside as exceptional the instance of bronchial aneurism, I must say that all the evidence I have been able to collect on this subject points to the pulmonary vessels as the usual source of hæmorrhage, slight and serious, and the only source of profuse hæmorrhage, except in cases of aneurism of the aortic circulation and of a general hæmorrhagic diathesis. It is possible that in cases of hæmorrhage springing from the walls of a cavity, the bronchial capillaries may contribute, especially in those instances where new vessels have been formed in cicatricial tissue, but in most cases (except the one of general hæmorrhage just alluded to), bronchial hæmorrhage is mixed with bronchial secretion, or if it springs from cavities with the secretion from the walls, and the result is a sanious mixture rather than a simple hæmorrhage.

Whether we consider the relative liability to rupture to which the pulmonary and bronchial vessels are exposed, by reason of their anatomical distribution, or the pathological evidence bearing upon the subject, the conclusion is forced upon us that when the rupture of blood vessels involves a considerable amount of bleeding, the pulmonary vessels are the chief sources of the blood; and although it may be true that the bronchial vessels only may be involved in bleedings of slight amount, if they are concerned in a copious hæmorrhage it is only as a part of the whole vascular system of the lungs, and in such cases the pulmonary vessels must still be credited with the largest contribution.

The distinction which has been drawn by some authors between the action of bronchorrhagia and pneumorrhagia is not supported by evidence, and the difference seems to be purely conjectural; nor am I aware of anything which lends colour to the theory. In this opinion I am not alone, and similar objections against the theory of bronchorrhagia have been urged by Ruehle, and by my colleagues, Dr. C. T. Williams and Dr. Douglas Powell, in their contributions to the literature of the subject.

CHAPTER III.

THE PATHOGENY OF PULMONARY HÆMORRHAGE.

THE pathological conditions which cause bleeding from the lungs are those which induce—

1. Alterations of the circulation of the lungs.
2. Diseases of the vascular walls.
3. Alteration of the blood.

1. The normal condition of the pulmonary circulation is altered, generally or partially, by influences tending to accelerate or retard the blood stream.

Hyperæmia, which is the simplest condition of increased supply of blood to the lungs, may result from increased muscular action: as, for example, when through activity of the individual the heart's action is accelerated and the blood is driven with more frequent stroke through the lungs; or it may result from nervous action, or from the effects of heat and cold, shock, and the like, or from the effects of irritants, from the inhalation of irritating air or the passage of irritating fluids over the bronchial tubes into the pulmonary tissue.

Under such influences as these, the blood accumulates in the vessels of the lungs, and, if the degree be slight, passes away and leaves no trace behind it; but in the more intense form it results in permanent plethora and a condition of stasis which is easily recognised after death.

Increased muscular action sometimes leads to rupture of a vessel immediately upon the strain, or to a congestion which is characterised by blood-spitting after some period has elapsed. Instances of death from excessive exertion are rare, and the following cases are given to indicate the pathological conditions which are brought about by such exertion.

The first two cases occurred under my observation, and are

recorded in the fifth volume of the 'St. George's Hospital Reports;' the other occurred in St. Bartholomew's Hospital, and was examined by Professor Kirkes.

Case I.—A man, aged 28, had been in good health, but after lifting some heavy weights he suddenly became very ill, sweating profusely, and being seized with acute pain in the region of the heart. Notwithstanding this, he went to work and continued at work up to five days before admission into hospital, when he became very much worse, suffering from vomiting and cardiac pain, and from blood-spitting. He died on the third day after admission.

On examination the lungs and all the organs were found much congested and full of dark blood. The heart was much dilated and the ventricles very thin.

Case II.—Another man, a labourer aged 23, was admitted into hospital in a condition of collapse resulting from prolonged and excessive exertion six days before. The man died the day after admission. In this case the collapse was so speedy that there was not sufficient power to raise blood. After death the lungs were found excessively congested and full of black blood, as were the other organs. Heart much dilated.

Case III.—A man, aged 25, had been suffering for some time from what had been called spasm of the heart: he had been relieved by the treatment adopted and was one afternoon after dinner walking with his wife, carrying a child in his arms. Suddenly he cried out that the old pain at his heart was coming on, and staggering into an adjoining shop, he died in a few moments.

In this case the structure of the heart was pale and the aorta atheromatous. Both lungs were found of a deep black colour from extreme congestion, but were healthy in structure. There was an irregular patch of a darker colour breaking into a grumous pulp under the finger, and gradually merging into the surrounding healthy but congested pulmonary structure.

In these cases the fatal result was induced by the failure of heart action; and to this condition must be attributed other cases of pulmonary congestion in which stasis arises simply from feebleness of the heart, due either to constitutional weakness or to imperfect growth of the heart, insufficient to maintain the circulation in a rapidly growing body.

The first condition, impaired muscular power of the heart,

may arise from simple debility, and occurs frequently in cases of anæmia, or it may be the result of fatty degeneration of the cardiac muscles.

The second condition occurs in young persons who grow rapidly, the heart apparently not keeping pace with the requirements of the body.

It is not at all uncommon to find in young persons, especially girls who have grown tall in a short time, symptoms of dyspnœa and palpitation on the slightest exertion, pointing to feebleness of the heart.

The following case, which is given by Andral in his 'Clinique Médicale,' may possibly be included in this category; unfortunately the condition of the heart is not given, but the initial symptoms point to difficulties of circulation.

Case IV.—A girl, 21 years of age, had felt for one year violent palpitations of the heart. Her breathing was habitually short, and she was put out of breath by the least exertion. Five days before death she spat up a considerable quantity of red blood, which recurred in greater quantities two days after, and continued to the time of death.

P. M.—The lungs were found peculiarly engorged with blood, and the engorgements which are carefully described were found in peculiar situations to which attention should be directed.

The summits of the middle portions of both lungs, and in the lung the anterior superior edge, were thus affected, and near the engorged nuclei were found several bronchial ramifications distended with black blood.

No description of the heart is given by Andral(p. 477).

It is worthy of note that no tubercles were discovered, although the lungs were evidently examined with the greatest care. The account of the case and the interpretations given by Andral of the post-mortem appearances seem to indicate some uncertainty in his mind as to the nature of the conditions which led to death.

He at first concludes that the expectorated blood proceeded from the entire surface of the bronchial mucous membrane, a conjecture which is not proved by the reported condition of the tubes, but he is in evident doubt on this point, because he goes on to say that perhaps the black and hard portions of the pul-

monary parenchyma indicated only those of the bronchial branches where the blood was more particularly accumulated.

The latter surmise with some probability seems to contain the real clue to the case: the localities affected are exactly those into which blood is generally driven by the reflux action of the inspiratory force, of which more evidence will be given in the next chapter; the accumulation of blood in special bronchia does not prove that the blood started from those tubes, but that it was finally landed there before death; and whatever be the correct view of the initial symptoms, it is probable that the special engorgements of the lung were due to blood which had regurgitated by insufflation into the parts described.

Feebleness of the right side of the heart is certainly a very influential agent in the production of stasis in the pulmonary circulation, and to this cause appears to be mainly due those cases of intense cyanosis which are the result, not of pulmonary disease alone, but to pulmonary disease and feeble action of the right side of the heart combined, for it is not until the powers of the heart are very much enfeebled that cyanosis occurs in these cases.

Many cases of blood-spitting that come under notice are preceded by great debility and depression, and it is probable that the bleeding occurs in consequence of a stasis resulting from the impaired power of the heart: cases of this kind improve rapidly under tonic treatment, and no recurrence of bleeding may happen.

Pneumonia is another condition which leads to streaky hæmoptysis, but the disease is so well known and has been so fully described that no further description is here needed.

Local alterations of the pulmonary and bronchial circulation lead to local congestions: the most important of which is that described by Laennec under the name of pulmonary apoplexy, and to which he ascribes those copious bleedings which are so difficult to control.

It seems evident, on reading Laennec's admirable account of this condition, that he assigned to this vascular rupture which, he knew, was by no means often accompanied by the presence of tubercle, the source of profuse hæmoptysis, and he attributed it simply to plethora, to the effects of prolonged heat

and excessive cold, and the suppression of habitual bleedings. He had known instances in which the quantity of blood raised had been enormous: in one case as much as ten pounds in forty-eight hours.

The condition induced has been frequently described: dark red patches are found in the lungs, generally situated in the middle of the inferior lobe of the right lung; the pulmonary tissue is soft and easily lacerated, and the air cells and alveolar tissue are impregnated with blood, which exudes in large quantity from a cut section. Sometimes considerable laceration takes place, and a clot of blood, dark and coagulated, is found in the middle of the patch. This form of congestion is often found with dilatation of the right side of the heart, and with enlargement and cirrhosis of the liver.

The following example may be quoted to show what appearances are found some years after an attack of this kind:—

Case V.—A man, age 29, under Dr. Powell, was examined in January 1878. Fourteen years before he had suffered from cough and bleeding from the lungs, and in 1873 he had a return of very copious bleeding.

Both lungs were found to have two or three long irregular bands or patches of deeply pigmented fibroid structure. These patches had been torn in the centre, and shreds of the same pigmented fibroid lung tissue remained. Here and there were some remains of old fibrine derived from previous blood-clots, some of which were calcareous.

The patches were evidently due to very old pulmonary apoplexies. They were found in the upper parts of the superior lobes, and the old clotted blood which had resulted from the apoplexy had in great measure cleared out, leaving many excavations of small size.

The liver was in an advanced condition of cirrhosis. The kidneys were small and hard. In all probability the case was one of alcoholism, leading to plethora and bleeding.

Engorgement of the lung results from pressure upon the central vessels arising from the presence of mediastinal tumours, carcinoma, lymphadenoma, and aneurysms.

The connection of hæmoptysis with cancer has been ably considered by Walshe, and he found that the source of hæmor-

rhage was evidently the vessels of the broken down cancerous mass, or of the pulmonary surface exposed by the decay and elimination of the cancer. In two cases exhalation from obstructed circulation was the cause of the blood-spitting, and in another case gangrene of the lung.

Mediastinal tumours produce pressure and obstruction leading to hæmoptysis. Lymphadenoma, not an infrequent form of these tumours, invades the lung tissue itself, and causes further encroachment on the vessels.

A peculiar form of engorgement of the lung, known as brown induration, is caused by the presence of mitral imperfections either by dilatation or by construction with rigidity. Under the influence of the back stroke of the heart the pulmonary veins become distended and enlarged, the capillaries being tortuous and varicose, and a hard brown condition of the lung results, in which extravasations of blood take place. This condition primarily affects the lung tissue in the region of the root, extending in an almost circular direction, with the root as a centre, so that the tract most indurated is the lower part of the upper lobe and the upper part of the lower lobe.

Congestion and irritation of the bronchial tubes, resulting from bronchitis and emphysema, frequently lead to bleeding, but the amount of blood raised is not large, and this condition is not to be confounded with the bronchorrhagia of Laennec, in which the bronchi are supposed to yield large quantities of blood: that profuse bleedings can take place from the bronchial capillaries only is very doubtful indeed, and I quite agree with Ruehle, who says that 'we are not justified in regarding the hæmorrhage, at least when it is copious, as coming from the bronchi. I have never yet seen any cases of the kind, and doubt their occurrence.' (Ziemssen's 'Cyclop.' vol. v. 527.)

I have already pointed out that this theory was evolved from the post-mortem staining of the bronchial tubes in cases of fatal bleeding: an appearance which is seen when the blood has started from distant sources.

A condition of irritation affecting the bronchial tubes is sometimes caused by the passage of irritating fluids, derived from cavities over the tubes, and to this I have alluded under the description of phthisis: generally, in cases in which the

blood starts from the tubes, the special secretion of the tubes is mixed with the blood. When the blood is unmixed it owes its presence to some disease affecting all the pulmonary vessels —the result of a general constitutional peculiarity.

The second class of pathological conditions which lead to hæmorrhage are those which concern the vascular walls. The alterations which these tissues undergo in phthisis and tubercle have engaged much attention, and many theories have been advanced to account for the bleedings which occur during the process of these diseases. To such great extent have these theories been carried that it would almost seem as if phthisis and tubercle monopolised pulmonary hæmorrhage, and it has even been suggested that if neither condition could be proved to be present in cases of copious bleeding a theory of latent tubercle would account for everything.

As long as the theory is maintained that all phthisis begins with an initial process of tubercle, so long must evidence be strained and theories started which must draw largely on the imagination, and it would appear as if pulmonary apoplexy which was credited by Laennec as the cause of copious bleedings even in the adult, had been almost lost sight of in the anxiety to connect pulmonary hæmorrhage with a previous condition of tubercle.

The whole question has been studied from the platform of Laennec, but, as the world has often seen, the tenets of the pupils of a great master are an expansion of his original views, and the result has been an extravagant exaggeration of his doctrines from which the master himself would recoil.

The term 'phthisis' is generally used to include several varieties of pulmonary disease which in their initial stages have little in common, but are ultimately linked together by terminating in general destruction of lung tissue; it is to this ultimate condition only, chiefly characterised by excavation, that I here attach the use of the word 'phthisis.' The vascular walls share with the rest of the pulmonary tissue in the process of destructive ulceration and softening, and the actual erosion which takes place from phthisical excavations in the vessels may be seen frequently with the naked eye in post-mortem examinations of such cases. We require no better proof as regards the

probable source of bleeding in some cases of hæmoptysis, and indeed, in cases in which the bleeding has proved fatal, the wounded vessel can often be identified; the marvel is that so few cases of fatal bleeding should occur, and that so many cases of phthisis should never show a sign of bleeding throughout the course of the disease. Protection is given by the time and by the special nature of the process; the vessels are more or less infiltrated by the products of inflammatory action, and if the disease is slow in progress the blood clots in the vessels and bleeding is prevented. But if the tissue which supports and shields the vessel be rapidly removed, or the ulcerative process cuts a vessel without time being given for defensive coagulation, that vessel with its altered walls may give way under but little pressure, and bleeding will result proportionate in quantity to the size of the artery.

On the other hand, as all observers agree, tubercular ulceration is essentially a slow process in which cellular growth invading the tissue and the blood-vessels plays a specially active part; induration of the environing parts is thus induced, and the vessels are completely blocked and obliterated, the necrosis which leads to excavation being consequent upon this absence of vessels.

Hence it follows that those forms of phthisis which are essentially tubercular are not characterised, as a rule, by hæmorrhage. This is particularly the case with scrofulous phthisis, a form of disease characterised by the accumulation of large cell-growths invading the pulmonary tissue and blocking out the pulmonary vessels.

The same thing is true even of the rapid form of phthisis, in which the tubercular process starts from several points, thus involving a considerable amount of lung tissue in a short time.

This form of phthisis is characterised by the invasion by cell-growths into the pulmonary tissue, and thus pulmonary bleeding is frequently prevented.

It must, however, be stated that this variety of phthisis leads to such firm and general adhesions of the pleura to the thoracic walls and diaphragm, and that the powers of the patient are so extremely enfeebled, that there is little capability for expectoration; and I have seen a lung converted by this process almost

completely into a bag, which showed at its base and in some portions of the existing lung tissue very evident traces that bleeding had taken place. In all cases in which the relics of bleedings can be detected, it is necessary to remember that the expectoration of blood will depend upon the powers of the individual, and upon the free action of the muscles of cough and expulsion, and the non-adhesion of the lung to the thorax and diaphragm.

In the syphilitic form of pulmonary disease, which is a non-tubercular process, there is a general and gradual induration, chiefly of the interlobular tissue, sometimes affecting considerable tracts of the lungs. In this form of disease blood-spitting frequently occurs, although the quantity of blood raised is not large. The individuals thus affected suffer from considerable dyspnœa, and the bleeding may occur in these cases from the bronchial capillaries, but I have been unable to establish this point. The induration which constitutes the initial stage of syphilitic lung disease breaks down very rapidly, and large cavities are formed, the clue to the initial stage of which is to be found in the tissue which lies outside the cavity, and gives the only indication of the previous condition.

It is possible that in some cases the capillaries may be weakened by a condition of amyloid degeneration, and to this some colour is lent by a case reported, by Dr. Wilson Fox, in the 'British and Foreign Medico-Chirurgical Review' for October 1865; but this must be considered as exceptional, for on looking over my cases of amyloid visceral disease, of which I have records, I do not find that blood-spitting is a frequent symptom.

The microscopical conditions of phthisis and tubercle have absorbed so much attention that it appears as if there was some risk of losing the broad features of the two processes derived from macroscopical examinations.

Two marked distinctions characterise as a rule the primary and secondary cavities in an ordinary case of phthisis; and by an ordinary case of phthisis I mean one of those in which the disease is developed rather rapidly in the upper lobe of one lung, which gradually liquefies, and its contents clear out, leaving a cavity which continues to secrete; the disease extends to

the inferior parts of the same lung, and to the upper lobes of the opposite lung. The whole course of the disease appears to me to be capable of explanation in the following way.

The initial disease may, as I have already intimated, start from a congestive catarrh, that is to say, there is an inflammatory condition in which the blood-vessels and the tissue are involved: the circulation in the upper lobe being more feeble than any other part of the lung, the function of this lobe being less active, the air cells being of large capacity, and lastly the respiration being due to a secondary current, the result of the expansion and compression of the lower part of the lung, stasis speedily results, and the exudation is profuse and liquid; a condition of softening soon follows, which may sometimes be seen in early conditions, in which the whole of the upper lobe becomes so soft and friable as to give way easily under the finger, and forms small cavities which show no thickening in the neighbourhood, and discharge from their softened sides a sanious fluid, consisting of *débris* of tissues, cells, blood-vessels, and blood.

The cavity continues to form and enlarge by the constant liquefaction and ulceration of its walls, and the secretion continues throughout to be stained with blood. The conditions under which such a cavity forms lie at the root of the ætiology of phthisis.

It would be out of place here to adduce all the evidence which has led to the idea that the respiration of the upper lobe is a secondary current, but some explanation may be given.

The expansion of the upper lobe in ordinary respiration is very slight indeed, the action of the draphragm alone being sufficient for the change of air in the upper lobe; if, as all observers agree, there is very little expansion of the upper lobe, and yet the air can be heard passing in and out of the upper lobe merely from the action of the diaphragm, it is difficult to see what other theory will account for this phenomenon except that to which I have alluded.

The same kind of secondary current is induced by this action as in the spray producers by the alternate expansion and compression of the india-rubber ball attached to one of the two glass tubes. There are many clinical phenomena of ausculta-

tion which appear to require such a theory: for example, a congestive catarrh may occupy the whole of the upper lobe, and the respiratory sounds give no indication of such a condition; the air enters into and out of the tubes, but it does not so mix with the secretions in the air sacs as to produce a crepitant roll. How then do we prove the condition? By making the patient cough the observer is often startled by hearing a general explosive rattling sound which indicates the condition, a most serious one, and one which is generally followed by rapid softness and excavation.

Now this absence of forcible ventilation induces a stagnant condition which is at least likely to encourage septic changes; moreover, the bronchial tubes of this lobe which are not altered in size remain permanently open, and do not filter the air in the same way as the bronchial tubes of the lower lung; consequently, it is probable that septic changes are more likely to take place. Added to this is the feebleness of circulation which undoubtedly characterises this lobe, and the tendency to hydropic degeneration of the vascular walls which follows stasis.

These conditions combined appear to me to make a strong case as to the predilection of the destructive process of phthisis for the upper lobe.

To return to the continued course of the case of phthisis. As long as the powers of the patient and the conditions of the lung in other parts remain unaltered—that is, as long as the muscular powers remain strong and the lung is not crippled by adhesion to the costal walls—the matter secreted is removed by cough; but after a time some of the sanious secretion remains behind, the next inspiratory effort lands some of it in an inferior tube, so that gradually it gets drawn into a communicating lobule, and there sets up irritation of tissue of a very chronic nature, in which the tissue is almost solely concerned; cell proliferation takes place, thickening and induration follow, and not only do the blood-vessels not share in producing the results, but they are actually precluded by the process from future complications. Hence the exudation from the vessels is prevented, and the tubercular abscess forms as a softened white product without the slightest stain of blood in it. This appears to me to afford a very decided, distinct, and unmistakable difference between the

primary cavity and the tubercular cavity which forms as the result of an irritation owing to the insufflation of non-ejected septic matter. How is it that the apex of the opposite lung is so soon and so generally affected? The answer to this is derived from a knowledge of the fact that phthisical patients almost invariably sleep on the sound side. Fortunately the decubitus prevents the inspiratory action of the thorax, but still some slight action is carried on, the fluid accumulates from the cavity, and gradually passes into and infects the opposite side.

This conclusion is arrived at by the careful consideration of the various localities which are first attacked, and from the evidence of many cases which proves that whereas the primary cavity is large, uniform, and single, the secondary cavities of the opposite upper lobe are of varied size according to their position and multiple.

That the primary cavity secretes a more virulent and septic fluid appears to depend upon the greater vitality and fluidity of the exudative material, whereas the softening tubercle is essentially necrotic and caseous.

In considering the distinction, then, between the process of phthisis and tubercle the main characteristic feature of the first is the share which the blood-vessels have from the beginning to the end of the process.

In the second they are exiled from one end to the other. The degree to which this exclusion is carried depends upon the condition of the process, not only as to kind but as to time.

In the course of phthisis bleedings of greater or less amount take place, of which we can only conjecture the source, the proximate cause of which may arise from mechanical strain on an injured vessel, and the necessity of taking adventitious circumstances into consideration as provocative of bleedings is shown by the evidence of all authors respecting the absence of hæmoptysis in a large number of cases of phthisis.

The form of phthisis in which the blood-vessels are found most exposed and pervious is that rather acute form of congestion which terminates in liquefaction. The result is seen after death in a large cavity, with vessels exposed, the walls of the vessels not thickened, the ends obtruncated, and often plugged with clot of blood more or less recent, the surrounding tissue

being not very dense and of a purplish red. The secretion from the cavity indicates the ulceration of the capillaries, which has been going on, and is of a reddish brown colour, due to the admixture of congested tissue and blood. In this form of cavity the initial condition has been a congestive catarrh, a catarrhal pneumonia, without indurative thickening, in which liquefaction has taken place rapidly, the lung tissue and the vessels sharing in the general destination. It frequently happens that blood-spitting has been observed some time before death in these cases, and sometimes death occurs from fatal rupture.

A remarkable case of fatal hæmoptysis in a child seven months old is recorded by Dr. Douglas Powell in the 'Pathological Transactions' (vol. xxv.). The child, who had been ill for three months with cough, was suddenly seized with a first and last attack of bleeding, expectorating more than half a pint of dark partially clotted blood. At the post-mortem examination it was found that death was due to rupture of a pulmonary artery traversing a cavity in the upper part of the lower lobe of the left lung. This cavity appeared to be due to the rapid breaking down of pneumonic consolidation.

A similar case is recorded by Dr. C. T. Williams in the joint work (by his father and himself), entitled 'Pulmonary Consumption.' The patient, a young woman, had been suffering from chronic basic pneumonia of two months' standing, when she was suddenly seized with fatal bleeding. In this case an aneurysm was found projecting into the cavity. Hertz remarks that he has seen profuse bleeding with caseous pneumonia in children three and four years old, and Lebert reports a case of fatal hæmorrhage from a cavity in a child two years old.

As a rule rupture of an undilated vessel takes place only when excavation has been rapid, the vessels being obtruncated, cut short off, or after having been exposed by rapid denudation they rupture from the removal of support and alteration of their wall. Such a condition occurs when the fibrinous remains of old blood residues, which will be fully described in the next chapter, liquefy and clear out, leaving the vessels exposed. Recently a case of this kind has come under my notice, the rupture of a small vessel having terminated fatally.

The formation of pulmonary aneurysm is due to a more chronic process: the vessel being generally imbedded in indurated tissue which has been removed at one part, and the vessel, thus denuded of support at one spot, and having undergone inflammatory changes depriving it of elasticity, yields at this point, bulges, and finally gives way. Dr. Powell, who has contributed an excellent paper on this subject, is also of opinion that aneurysms generally form in old standing cavities, and this quite accords with my own experience; but I am inclined to think that old standing cavities which are undergoing some new and rapid change are those which aneurysm particularly affects. In two or three instances I have come across small aneurysms developed in small cavities which have been formed by the evacuation of blood residues, and in these instances it is evident that there has been, first, a chronic condition of thickening involving the tissue in the vicinity, and subsequent rapid softening from the liquefaction of these nodules.

In one case the aneurysm had been healed by the inflammation of the coat and thrombosing of the blood within the blood-vessel at its proximate portion, this inflammatory process being evidently the result of the alterations in the fibrinous nodule in which the aneurysm was situated.

These aneurysms undoubtedly are the most usual cause of fatal bleeding from the lungs; they are to be found of various sizes, from a hemp-seed to a Maltese orange; they occur at all ages, and depend upon a local condition, and are in no measure related to systemic aneurysms. A case has been recorded by Dr. Hilton Fagge, the patient being not quite three years old, but the age at which they usually occur is between twenty and thirty.

The best method for detecting them is the following:—The lungs should be both removed, together with the trachea, and the air passages first carefully examined to see from which side the hæmorrhage has proceeded. Frequently this cannot be told by the condition of the clot, inasmuch as the largest amount of clot is sometimes found in the most active lung. If a large cavity occupies any portion of the lung, it must be carefully searched, as the aneurysm is generally found in the primary cavity. Palpation should then be made carefully over

the surface of the lung, and any nodular swelling should be cut across. I have thus succeeded in detecting very small aneurysms which would otherwise have been probably missed: it is necessary to remember that aneurysms are often multiple, as many as four have been found in one lung—a condition recorded in Rasmüssen's paper on the subject. If the aneurysm is not detected in the cavities, the bronchial tubes must be then carefully examined.

This form of arterial disease was fully recognised and described by Rokitansky in his 'Pathological Anatomy.'

Pulmonary aneurysm may also form and perforate a bronchial tube, and thus lead to fatal bleeding. I have come across two instances of this kind, both in connection, apparently, with the formation and subsequent removal of old fibrinous residues. The following case I owe to the kindness of Dr. Powell:—

Case VI.—Elizabeth L——, age 30. Previous diseases: scarlet fever with dropsy, measles, and rheumatic fever. Family history not certain. Two years before admission in September 1878, had suffered from indigestion, and cough began in the October previous. She had been attacked with hæmoptysis, which was reported to be slight, in July. Diarrhœa nil.

When she was admitted, physical signs were found indicating the presence of a cavity in the left upper lobe. She improved under treatment, and gained weight, and was going out, when, on February 9, 1879, she was suddenly seized with hæmoptysis, and raised a pint of dark blood. She became very pale, struggled much as if fighting for breath, and died in a very short time, about fifteen minutes from the time of seizure.

Sectro Cadaveris.—Body well-made and stout: none of the usual appearance of phthisis. Much fat on the abdominal walls.

Left lung adherent in front, and behind at the upper lobe. In the middle of this lobe a cavity was found the size of a small Maltese orange, with a thick white enveloping capsule a quarter of an inch thick. This cavity contained some curdy soft *débris*, which were totally different from the secretion from the ordinary cavities found in phthisis. It contained a small quantity of black blood, which had been poured out from the aneurysm situated between the cavity and the root of the lung. On slitting open the communicating bronchus, a small aneurysm was detected, not

larger than a hemp-seed, which had perforated the bronchial tube. This aneurysm sprang from a secondary branch of the pulmonary artery, which beyond the aneurysm was completely occluded by an adherent thrombus. This thrombus was close to the circumference of the cavity by which it had been pressed. The main residual force of the artery had acted straight in the direction of the aneurysmal sac, and the wall of the vessel was diseased.

In the upper axillary portion of the lower lobe was found a hard white round fibrinous nodule, which on section was found still surrounded with blood, the size of a finger-nail, and below this was a cavity similar in all respects to the cavity in the upper lobe, except that it was half the size and contained near the periphery the remains of a circular fibrinous nodular mass, which was softening, and had evidently formed the primary basis before the excavation had taken place.

A number of round hard nodules of the same character and size were found in the middle portion of the lung, but no tubercle was detected by the naked eye.

The right lung was much dilated, and flecked with large blotches of inhaled blood, and all the tubes were full of dark blood.

The condition of the base of the lower lobe is depicted in Plate I.

There was no disease of the other organs.

The cavity found in the upper lobe of the left lung was of a very peculiar nature: it was made, as far as I could form an opinion, by the breaking down of fibrinous matter which had in all probability been placed there in the course of a previous hæmorrhage. This opinion receives considerable confirmation from the presence of the other cavity of smaller size in the upper part of the inferior lobe situated in the mid-axillary region close under the periphery. This cavity could be distinctly traced to its primary starting point, a large portion of hard fibrinous nodules still remaining. This nodule was identical in form and appearance with one situated above it, and which was still surrounded, and blood (in a ring) which had lost some of its colour. I thought at first that there was no tubercle at all in the lung, and there was none that I could detect with the naked eye, but when the other lung was more carefully examined, one or two solitary tubercular nodules were found in the right lung.

The aneurysm which had made its way into the bronchial tube was not larger than a hemp-seed, and was formed close to a clot which closed the rest of the artery, and had been formed in consequence of the proximity of the hæmorrhagic nodule.

The other case in which I found an aneurysm in a dilated bronchial tube was examined in 1876. History showed that the patient had suffered from frequent hæmoptysis, from a pint to a quart, up to the day of death.

In this instance the aneurysm was no bigger than a mustard-seed.

Blood-spitting may also result from the invasion of an aortic aneurysmal sac into the lung: the blood oozing from the sac into the bronchia.

Such a condition may occur in aneurysm of the posterior portion of the transverse or descending portion of the aorta, or it may occur, as in a case given by Dr. Stokes, in the abdominal aorta, the aneurysm passing upwards into the lower lobe of the left lung, the perforation of the aorta being on its anterior wall.

Aneurysm of the bronchial artery has been known to result in fatal bleeding, and such a case has been recorded by Dr. Church in the 'Pathological Transactions.' Up to this time I have not seen an example of this form of aneurysm.

Thrombosis and embolism are conditions which lead sometimes to bleeding from the lungs.

Thrombus often occludes the branches of the pulmonary artery, occasionally the main vessel, and it results from stagnation and coagulation of the blood, which may be caused by disease of the arterial wall, or by sluggish circulation consequent upon an enfeebled muscular power of the right side of the heart.

The pathology of thrombus and embolism has been so fully described that it requires little description here, but there are some points with reference to the agency of pulmonary disease in causing thrombosis that require a passing remark.

When a thrombus occurs in a large branch of the pulmonary artery, the condition induced is one of anæmia, and the portion of lung from which the functional supply of blood is thus cut off becomes distended and pale, closely resembling emphysema in appearance. A very great strain is thrown upon the rest of the pulmonary arterial tract, which is shown by a remarkable distension of the artery of the same side, and sometimes by extravasation of the terminal arterioles. The amount of blood-spitting, however (even in a case in which the main right artery is blocked), is very slight.

Blocking of the functional artery causes collapse of the distal portions from the pressure of the atmosphere upon the vascular walls; hence the formation of the wedge-shaped block is sometimes prevented.

Moreover, this wedge-shaped block is formed irregularly in cases of small descending branches of the pulmonary arteries, from the fact that the blood, as it passes out of the capillaries in the fashion described by Cohnheim, is inhaled into contiguous alveoli, and here becomes impacted, gradually losing colour.

The wedge-shaped block occurs generally in the middle periphery of the lung, and from embolus of the bronchial arterioles, the result of mitral fringing.

Thrombus occurs as the result of a contiguous secreting cavity, the resultant secretion soaking into and producing inflammation of the arterial walls. It also has a tendency to occur by a similar process in those cases in which there is considerable feebleness of vitality, in consequence of which circulation is retarded, and secretions which ought to be expectorated tend to accumulate by gravitation towards the inferior parts of the lung.

Bleeding must not, however, be considered as a constant result of thrombus. In a case observed with great care, and reported in one of the medical journals by Dr. Powell, no hæmorrhage occurred from the time of seizure, and this symptom is not infrequently absent.

That bleeding should not follow the obstruction of a large vessel is a most cogent argument against the theory of collateral hyperæmia from the presence of tubercle as a cause of hæmorrhage.

Bleeding may occur in phthisis from old cavities in which fresh vessels have been formed in new tissue during the process of healing. If fresh inflammation occurs in these cavities, these new formed vessels rapidly give way, and give rise to bloody sputa, which, when mixed with the old pigmented tissue presents an appearance similar to prune juice. Sometimes cavities undergoing this process are seen in a lurid erysipelatous condition, lined with a soft pyogenic membrane.

The secretion from a gangrenous cavity is sometimes in like manner mixed with blood derived from capillary sources and the

bronchial tubes, over which the secretion pours are seen stained of a deep claret colour; the same secretion passes over into the opposite lung and stains it with purple patches.

The irritating secretion from a freely secreting cavity pouring over the tubes may induce such a condition as to lead to bleeding, the blood mixing with the secretion. A remarkable instance of this occurred under my observation which is worth noting:

Case VII.—A right lung was examined in which a large freely secreting cavity was situated at the upper lobe. The lower part of the lung had been prevented from free action by the raised condition of the diaphragm, which had been forced upwards by an increase in the size of the liver due to amyloid deposit. The bronchial tubes of the middle part of the lung were found full of secretion mixed with blood, the secretion being the same otherwise as that from the cavity, but the tubes of the lower part were quite transparent and healthy, the fluid had not been drawn over them.

Under whatever aspect tubercle is considered, the main characteristic of the process is the infiltration of cell-growths into the alveolar tissue, and the consequent compression and obliteration of the blood-vessels. It is essentially in the adult a chronic process, the duration of time required for the development of tubercle being in some measure proportionate to the period of life at which it is formed.

All observers are agreed that the essential feature of the process is the cellular invasion, which throws out, as it were, an outwork, in which the blood-vessels are so blocked and obliterated that no blood can be driven into the part affected, and all attempts at injecting tubercle have signally failed. This is so universally the case with tubercle that it is difficult to understand how hæmorrhage can be attributed to tubercle in what is termed its crude stage. Indeed it is apparent that this difficulty has been appreciated in some quarters because recourse is had to the surrounding congestion, to the collateral strain on the blood-vessels which follow in the train of tubercle.

Whatever satisfaction may accrue to the supporters of this theory from the admission that traces of blood might in minute quantities result from these conditions, neither assumption is sufficient in the remotest degree to account for those copious

bleedings that take place often as an initial symptom, sometimes without evidence of pulmonary disease.

The connection between pulmonary hæmorrhage and tubercle stands on no pathological proof, and it is impossible to examine many tubercular lungs without coming to the conclusion that tubercle, instead of fostering bleeding, actually prevents and discourages it. Both lungs may be stuffed closely with tubercle, as in acute tuberculosis, and no bleeding can be detected; and even in cases where the infection is more local, and firm and compact masses are found invading large tracts of the lungs, yet no bleeding occurs.

The same thing is true, as I have already remarked, of scrofulous phthisis and acute phthisis, both of which forms are tubercular in character.

Congestion may be found surrounding tubercle, but I have never detected any hæmorrhage in the part affected, and as regards the supposed effect of a collateral hyperæmia, that is simply a conjecture which cannot be proved, and against which theory the following arguments may be raised.

The pulmonary vessels are capable of undergoing considerable strain without rupture; bleeding does not occur even when one lung has collapsed from pneumo-thorax, which, under whatever circumstances it occurs as regards the previous condition of the patient, certainly results in throwing a considerable quantity of blood on the visceral organs. This is a fact derived from the experience of many examinations, in which the liver, spleen, and kidneys were found full of blood when one lung has been perforated, and has been followed by speedy death.

Bleeding does not always occur even when there is thrombosis of a vessel of considerable size; the strain in the pulmonary artery may be shown by a considerable dilatation, but it does not always rupture. The whole of a main branch may be thus completely plugged, and only streaky hæmoptysis of a transient nature be observed. Moreover, the formation of tubercles is slow, and occupies generally but small portions of the lung.

The only way in which I can satisfy myself that tubercle may result in pulmonary hæmorrhage is this, and examples of the kind may be sometimes seen in the post-mortem room.

Tubercle may in its formation compress and stop a portion

of a small pulmonary vein (it must be a small one), nipping it and obstructing the current in the capillaries; in this way the pulmonary vein becoming blocked, it is possible that a slight capillary hæmorrhage might follow, although I have seen no instance of the actual rupture, and the usual result of this blocking would seem to be a simple stasis resulting in streaks of black pigment. Probably Rindfleisch had such an instance in his mind when he said this: 'If one of the smaller branches of the pulmonary artery is involved in tubercle granule, and occluded, the arteries which remain open must contain more blood, be subjected to greater pressure, and any point in their walls is likely to rupture.' Hereby having recourse to the theory of collateral hyperæmia, which must be very considerable indeed to produce bleeding.

After some experience in the examination of tubercular and non-tubercular lungs, I must express my opinion that bleeding from the lungs appears to stand in no relation to the amount of tubercle, and is independent of it; but the presence of tubercle in large quantities, whether it be in the scrofulous lung, or in any other form, would suggest to me the probability that the patient had not been subject to any copious attacks of bleeding during the time of the tubercular process.

Nor does the theory of tubercular ulceration, as causative of bleeding, stand in any other position, inasmuch as the liquefaction results from a necrosis due to absence of blood-supply; hence the contents of a strictly tubercular cavity are not found mixed with blood, and it cannot be conceded that such ulceration, when of a tubercular character, has encountered any blood-vessels which could bleed. Occasionally ulceration of a non-tubercular kind is seen excavating tissue from around a tubercle, but it is non-tubercular, inasmuch as the tubercle may remain like an islet in the middle.

From all the evidence I have been able to obtain on this point, tubercle seems to have been very unjustly accredited with hæmorrhage, nor has any argument or evidence been brought forward to show its connection with those copious bleedings which are so often brought under notice.

Having now pointed out the relation of pulmonary hæmorrhage to the processes of phthisis and tubercle, I shall proceed

to describe the conditions which establish a proclivity to bleeding; and attention must be directed first to the phenomena of that peculiar tendency to general bleeding which is now known by the name 'hæmophilia.' In doing so I shall draw upon Dr. Legg's careful treatise on the subject, in order that by making excerpts from an unbiassed account of this general disease, I may make comparison with the phenomena which accompany a more local tendency, which constitutes that form of disease to which I believe initial hæmoptysis must be attributed.

Hæmophilia is a constitutional disease which is congenital and distinctly hereditary; it begins with infancy, it ends with the patient's life, and it is characterised by general and abundant bleedings, and by swelling of the joints of a peculiar kind. It attacks males much more frequently than females, and the disease affects the latter sex with far less intensity; it is the result of inheritance derived from parents or grandparents, and is found in cousins. The subjects of hæmophilia are stated by some observers to be intellectual; their skin is thin and transparent, the veins showing plainly, but generally they look well and healthy.

There are three degrees of intensity in which the disease may occur: in the highest degree there is a marked disposition to bleedings of all kinds, and especially to swellings of the joints.

In the second degree 'the disease,' to quote Dr. Legg's words, 'is infinitely less intense': spontaneous hæmorrhages from the mucous membranes only are present. Neither traumatic hæmorrhages nor ecchymoses are met with, and the joint affection is absent, or represented only by rheumatic pains.

In the third degree there is a tendency to spontaneous ecchymoses, but when affecting women menstruation is early and abundant.

As this is a congenital disease it is not to be expected that bleeding from the lungs, which symptom belongs to the adult age, will frequently occur, and in the numbers quoted from Grandidier in only 15 out of 256 cases did bleeding proceed from the lungs.

To quote Dr. Legg again: in childhood these persons more frequently bleed from the nose than any other part; after puberty, bleeding from the nose and mouth becomes less common, and is replaced very often by hæmaturia or by bleedings from the bowel; vomiting of blood and hæmoptysis become more common.

Lastly, I must point out some of the macroscopical appearances of the viscera as observed after death. Sometimes the only thing noted has been a marked anæmia of all the organs, a condition dependent upon the loss of blood; but in other cases the heart has been found pale and sometimes fatty, the right side being thin and dilated, and the left hypertrophied, while the vessels appeared thin, flaccid, and transparent. The liver has also been noted as enlarged and fatty—a condition which belongs to other diseases and cannot be therefore considered as characteristic of this form only.

Since Dr. Legg's treatise was published, the pathology of the disease has been further elucidated in an excellent paper, by Dr. Percy Kidd, in vol. lxi. of the 'Transactions of the Medical and Chirurgical Society,' in which the altered conditions of the vascular walls is described with much care.

The specimens were taken from a child six years of age, and the following is the account given:

The lungs were remarkably bloodless, as were also the liver, spleen, and kidneys. The left ventricle of the heart was decidedly fatty; valves natural. A very pale clot was interlaced in the columns of the right ventricle and auricle; the remaining blood looked almost like water. The arteries, capillaries, and veins all showed considerable alterations. The endothelial cells lining the vessels had undergone an extensive proliferation, in some so considerable as to block up the vessels more or less. The muscular coats of the arteries were altered: the walls being much thickened without any distinct muscular elements, apparently resulting from a hydropic degeneration of the muscular fibres.

It would seem then that this disease is due to structural imperfections of the vascular walls, the result of hereditary transmission. With the points regarding this disease which have just been dwelt upon before the view, let me pass on to

the consideration of a case of initial hæmoptysis of a remarkable kind.

Case VIII.—Charles T——, age 22, under Dr. Symes Thompson. The family history as far as it extended was this: Both father and mother were alive and healthy, and out of their family of eight, two daughters had died when young, the others, with the exception of the patient, being healthy. The patient had suffered from rheumatism.

Blood-spitting occurred first in December 1876, and took place previous to cough. He spat up at first half a pint of blood. In March he spat up more blood, which recurred in three weeks' time and again in nine weeks' time, at which latter period he raised as much as a quart. Loss of flesh lately very great, with night sweats and cough.

He was admitted into hospital on August 2, 1877, and died October 17. During the time he was in the hospital he had several attacks of copious bleeding from the lungs, the nose, and the bowels. He died of exhaustion from loss of blood.

P. M.—Body emaciated and pale, lips white. Right lung adherent at the upper part: the whole lung was much congested, the pulmonary tissue and the bronchia throughout stained of a deep purple colour, but it was noted that the descending tubes were stained more deeply than the others. The upper lobe was excavated by multilocular cavities close to the periphery of the lung, and appeared to be terminal to the tubes. The bronchia were much dilated and pouched in places. The lung tissue was studded below with grey tubercle of the arborescent form. Left lung firmly adherent at apex. Puckering at the back of the apex, corresponding to which surface depression was a small contracted cavity, with hard tissue round it not larger than a nut close to the summit. There were small lurid cavities of a similar kind in the upper part of the lower lobe, and arborescent tubercle in the lower part of this lobe, with small yellow rounded patches the size of a millet-seed scattered about here and there.

The heart was large: the valves were healthy, but the muscular tissue was pale. The right side of the heart was dilated, the left hypertrophied. Weight of heart $13\frac{1}{2}$ oz. The following measurements in inches were taken of the orifices: aorta, $2\frac{1}{4}$; mitral, 4; pulmonary, $3\frac{1}{2}$; tricuspid, 5. The vessels of the bronchial tubes attracted observation from their dilatation and apparent irregular distention; the pulmonary tissue was also peculiarly vascular, and not affected simply with congestion. The liver was large and fatty,

weighing 5 lbs. 6 oz., and the spleen was large. The kidneys were natural.

The case affords an example of initial hæmoptysis; and I think it will be at once evident that there are many points which appear to form connecting links between this form of disease and that which has been just described, especially as it occurs in the second degree of intensity.

The hereditary history of the case is defective, as it ought to have included the grandparents; in a subsequent chapter I shall endeavour to show how hereditary transmission can be traced in cases of initial blood-spitting.

The cavities which were found in the lungs are peculiar, and were secondary to the results of previous hæmorrhage, formed by the evacuation of fibrinous residues, which will be more fully described in the next chapter. The tubercle was of recent growth, secondary to the cavities, and cannot be with any probability credited with causing the bleeding. The conditions of the lung, and especially of the bronchia, attracted my attention by the peculiar character of the vessels, which appeared abnormally dilated. The condition of the heart pointed to some vascular impediment, and it must be remarked that the weight of the heart in this case is greater than it would have been from phthisis only, the extreme weight given by Peacock for this form of disease being only 11 oz., the average in males being 9½ oz. The liver was enlarged and fatty, and the spleen large.

The conditions during life and after death are so similar to those which have been quoted as characteristic of hæmophilia, that it appears to me difficult to avoid the conclusion that the two forms of disease are essentially the same. It is rare to meet with cases of this kind in which the post-mortem appearances are not greatly complicated by subsequent events, and I have no other case which so directly indicates the conditions which ought to be looked for in cases of profuse initial bleeding. The connection between this disease and hæmophilia will be again considered from another aspect.

Another case may be cited for the indications of disease which appeared to have caused the primary bleeding, but it is complicated by a cirrhosed condition of the liver, and is not so positively connected as the previous case with hæmophilia.

Case IX.—Henry S——, aged 28, under Dr. Powell. Father had been subject to phthisis, and died from rupture of a blood-vessel.

The patient had enjoyed good health until seven years before his death, when he interfered in a fight and was hurt. Hæmoptysis followed which lasted for several days, the quantity was not very great. Ten months before admission into hospital he appeared to have had an attack of hæmatemesis, and two months afterwards he began to suffer from cough, losing flesh.

He was admitted on December 10, and on the 15th he was suddenly seized with bleeding after dinner which proved fatal.

The lungs were found generally very soft and blood-stained: the upper lobe of the right lung was densely pigmented, and at the summit close under the periphery were a number of small blood-stained clean-cut ovoid cavities: the lung being puckered in above each cavity, and much pigmented in the neighbourhood. A large cavity, as big as a Maltese orange, occupied the central portion of the upper lobe, and in it was a vessel traversing the cavity which was ruptured the sixteenth of an inch in diameter. The rest of the lung was pigmented with streaks of black, evidently from an old condition of disease. Recent grey transparent tubercle was found in the lung. The left lung was in a similar condition as regards the general pigmentation, the small peripheral cavities, and tubercle. The heart weighed 9 oz., it was hypertrophied. The liver was cirrhosed. The spleen was large and much ecchymosed with extravasated blood: weight $14\frac{1}{4}$ oz.

The general pigmented condition of the lung points to the original cause of bleeding as proceeding from some vascular condition affecting the lungs generally: as in the previous case the small cavities, and in much probability the large one, were due to liquefaction and evacuation of hæmorrhagic residues. A relic of former bleedings was found in the left lung in the shape of a large fibrinous patch, which still retained the colour of blood.

On looking through the records of cases which I have examined, I should say that indication of previous tendency to bleeding is given by the general condition and colour of the pulmonary tissue, which will vary according to the duration of the tendency. If of recent date the pulmonary tissue is found much blood-stained of a more or less purple colour; if of longer duration, the black pigment of old blood is found interspersed among the pulmonary tissue; and I conclude from these

and other points of evidence that profuse initial bleedings are due to some vascular condition general throughout the lungs, and not to an incipient phthisis or an early formation of tubercle.

In continuation of this subject, I shall proceed to consider how a condition closely allied in some respects to hæmophilia may be artificially induced, and this will bring me to the third class of conditions, alterations of the blood itself.

The addition of fluids to the blood through the stomach increases the intravascular pressure, and remarkable instances of the effects of copious potations are to be seen in beer-drinkers, cellarmen, and others who imbibe large quantities of beer or light wines.

But the increased amount of fluids not only swells the vessels, but it increases the work of the heart, and eventually a hydropic degeneration of the tissues generally is brought about, which in some cases induces a general condition which resembles hæmophilia.

Case X.—Henry H——, aged 23 years. Mother died of cough. Father healthy.

Has been a cellarman for eight years, and accustomed to drink beer in large quantity, no spirits. He was in a condition of marked alcoholism. Face very congested and in a general state of purpura; gums very spongy, and bleeding. He had a pimple on his nose, which when scratched would bleed for an hour. Has passed, two months ago, blood in the urine. Urine not albuminous. Hæmoptysis, one pint three months ago, after lifting a barrel. Used to weigh 12 stone, now weighs 10 stone 9 lbs.

This man was in an extremely shaky cachectic condition.

Physical symptoms.—Marked retraction of lung under right clavicle, which I believe was due to contracting hæmorrhagic nodule. On the left side were moist sounds of œdema.

This man was treated with iron and cod-oil, and rapidly improved; he reduced his drink, went out in the open air, became steady, and lost all the look of purpura, and gained 7 lbs. in weight in two months. The sounds of crepitation disappeared, and no appearance of blood recurred.

The following fatal case of this character is taken from Flint's work on 'Phthisis':

Case XI.—A corpulent man, and an habitual beer-drinker, had been ill for several days with stitch-like pains in the chest. He was suddenly seized with hæmoptysis, and died in a few moments. He raised nearly two quarts of blood, and a large quantity was also found after death in the stomach.

The lungs in this case were found to be intensely congested.

In a note appended to the account of the case, Flint conjectures that the case might have been one of gastrorrhagia, because blood was found in the stomach: this is a quite probable, but not of necessity the cause of death, inasmuch as the blood might have been simply swallowed; whatever doubt may be raised on this point it will not alter the condition of the lungs, for which the case is here quoted.

These cases occur frequently among our hospital out-patients, and bleeding from the lungs is to be attributed to this cause, the amount of blood lost being sometimes very large. The appearance of the organs is very characteristic of the disease.

All the organs are found large and full of watery blood; the lungs are soft and friable, streaked with pigment, and much congested—adhesions are common; the heart is large and dilated; the muscular structure sodden, flabby, and fatty; the liver is congested and fatty; the spleen is very much enlarged, and sometimes almost diffluent; the kidneys are coarse, congested, and large, the capsules adherent, and the surface roughened.

This condition is encouraged by want of exercise and absence of fresh air, and consequently especially affects cellarmen, barmen, and barmaids, and women who take little exercise and much liquor. It is to this that many cases of plethora occurring among females are to be attributed, in whom, without any evident pulmonary disease, profuse bleedings occur.

A condition of plethora in less degree is induced in florid women in whom the catamenia have been from some cause or other checked temporarily.

Finally, bleeding results from those forms of disease in which the blood is altered, as in scurvy, purpura, and the various eruptive fevers.

Twenty-three cases of fatal bleeding have come under my

observation, and in five of these the injured vessel was not discovered; of the remaining eighteen cases in thirteen the rupture of a pulmonary aneurysm was established, six in the right lung, seven in the left. In one case a ruptured vessel without aneurysmal dilatation was discovered, and in one case the fatal bleeding proceeded from the pulmonary and bronchial capillaries. In one other case a pulmonary aneurysm which had not burst was detected. In one case death was caused by the rupture of an aortic aneurysm into a bronchus, and in one the blood came from a small aneurysm in the coronary artery of the stomach, the result of ulceration.

Out of the twenty-three cases of fatal bleeding, twenty occurred in men, three only in women. Pulmonary aneurysm was only found in two female cases.

CHAPTER IV.

THE PATHOLOGICAL TRACES OF HÆMORRHAGE.

The fact being established that pulmonary hæmorrhage may proceed from the rupture of a single vessel, the exact position of which may be identified after the death of the patient, we are in a position to investigate the results of such a rupture.

Before this fact was known, observers were placed at a disadvantage when they had to examine lungs after death from a fatal bleeding, inasmuch as they were not in a position to determine how much of the appearances in the lungs were due to primary causes, how much to secondary results. When a profuse bleeding has been the cause of death, blood is found in several localities, even in the lung opposite to that in which the fatal rupture has taken place, evidently planted in those regions by secondary influences. It was this very engorgement of different parts of the lungs which led to the erroneous conclusion that the bronchial vessels were the frequent source of the blood; as the appearance of the lungs in these cases is extremely important, three cases shall be given to show exactly what takes place.

The first case is one in which there was no disease affecting the pulmonary tissue; death occurred from the rapid oozing of blood from a small opening in an aortic aneurysm into the left bronchus.

Case XII.—A man, aged 37, was under my care during a period while I was taking the wards of my colleague Dr. Symes Thompson. He had been suffering from symptoms of pressure on the left bronchus, with frequent blood-spitting.

The physical signs showed compression of the left bronchus, with moist sounds at apex and base, which were conjectured to be caused by blood oozing from an aneurysm. The right lung was

much distended and perfectly healthy. The man was seized with a sudden attack of copious bleeding while at the closet, which proved fatal after continuing for the space of an hour.

P. M.—An aneurysm was found proceeding from the under part of the arch of the aorta, and opening into the left bronchus by a small aperture; the aneurysm had so encroached on the lumen of the bronchus as to partially close it.

The right lung was found very much distended, and overlapped the mesosternal line and the heart; on section it was found completely gorged with blood from apex to base, the lobules being full, distended, and having a dappled appearance, the centres very dark, the outer parts lighter in colour. The bronchi were full of blood.

The left lung was much smaller: dark blood was found in a band at the summit of the lung, also at the base, and more recent clotting was found in the bronchial tubes, as well as also at the periphery in the middle axillary region.

By the obstruction of the left bronchus, the right lung had been exerted to its full extent, and the extraordinary suction power of the lung was shown by the complete engorgement of the pulmonary tissue with blood derived from the bronchus of the left lung; sufficient breathing-space was afforded by the left lung to support life until the right lung was completely full.

The power of insufflation in the left lung is shown by the presence of blood in the upper lobe and other parts. From this it is quite evident that gravity is not the only force which influences material substances found in the lungs, and indeed gravity cannot act unless air can make its exit from the lung; the inspiratory force can act independently of gravity. Gravity does not produce any apparent effect if opposed by air in the lungs.

The next case is one in which the quantity of blood ejected was small, but the fatal termination was more rapid; the respiratory powers being thus shortened it is to be expected that the positions assumed by the regurgitant blood will depend more upon the influence of gravity, which would be effective after the death of the patient.

Case XIII.—Henry W——, aged 20 (under Dr. Powell), had been subject to cough for ten months, and during that time had only been once attacked by blood-spitting which had streaked the sputa.

Two months after admission he was walking about after dinner when blood spurted out of his mouth and nose (about 6 oz. in all), and he died in ten minutes.

P. M.—The right lung was firmly and generally adherent to the chest wall. The upper lobe was excavated and contained two small aneurysms the size of a pea, one of which had given way. The rest of the lung was solidified. A careful examination of the left lung showed a very large recent clot in the main bronchus, extending mainly into the descending branches, but the ascending branch also contained a small clot. The upper lobe was in the middle and back portions stained with blood: more stain was found in the middle peripheral region and in the inferior lobe.

The left lung was slightly adherent.

In this case the power of insufflation was materially diminished by the condition of disease in the lungs: one side was completely adherent and solidified, thus preventing much of the action of the other side; consequently, the positions assumed by the regurgitant blood were chiefly due to the influence of gravity, although this could not act unless the lung had been previously exhausted of air.

The third case shows the peculiar power of insufflation in affecting the most expansive parts of the lung.

Case XIV.—A young man (under Dr. Symes Thompson) was seized with copious bleeding from the mouth, and died in fifteen to twenty minutes, the quantity of blood ejected being a pint and a half.

P. M.—A cavity was formed in the upper lobe of the left lung, traversed by a trabecula formed by a pervious vessel, one side of which showed a recent blood-stained rent. This lung was generally distended in the lower parts, and was partially adherent. The right lung was very much distended and extremely pale in texture, and in marked contrast to this general condition were flecks of black blood, which were found generally under the periphery all round the lung.

In the upper lobe, patches the size of a thumb-nail were found in the central part, and the other localities especially affected were the middle axillary portion and the anterior inferior border. Much smaller patches of blood were found in this part. The bronchia were full of clot. A quantity of blood was also found in the stomach, but this viscus was quite healthy.

On the opposite page I have given two drawings taken from two cases of fatal hæmoptysis. It is rare to have such opportunities as were afforded by these cases for observing the morbid appearances of blood inhaled by a lung unaffected with phthisis.

Fig. 1 represents the condition of the upper lobe of the right lung in Case XIV. The source of hæmorrhage was a ruptured vessel in the left lung, and the plate shows the power of inspiration in forcing blood into the upper lobe of the opposite lung against the action of gravity.

If the fatal hæmoptysis had occurred in the case of a woman the amount of blood inhaled would have been larger, owing to the peculiar sexual differences of respiration.

Fig. 2 represents the lower portion of the right lung in the case of Elizabeth L., recorded at page 27. It shows the tendency of the blood to be drawn into those parts of the lung, the peripheral border, and especially that part of the lower border of the inferior lobe which corresponds to the summit of the diaphragmatic arch.

If the fatal event had occurred in a man the amount of blood inhaled into this lower border would have been larger.

In this case also the hæmorrhage had taken place from a ruptured pulmonary aneurysm in the opposite lung. Death ensued not from suffocation but from the rapid depletion of blood from the lungs; the heart was found firmly contracted, but both ventricles were completely empty.

These cases sufficiently indicate that the appearances which are found after death from pulmonary hæmorrhage are due to the insufflation of blood into the lungs while the bleeding is going on.

The bronchia are invariably found full of clot, and their mucous membrane stained more or less according to the time after death at which the examination is made; and it is often impossible by simple inspection of the main bronchus of either side to determine from the amount of clot the lung from which the blood has started.

It is evident then that the presence of blood in the air passages is no proof that the blood originated from them: it simply points to the fact, that the blood is landed in them when the powers of life are failing, or by the spasmodic gasps made during

the dyspnœa arising from the continued bleeding stopping the supply of air, and that the blood remains in the bronchial tubes when death ensues. The same insufflation of blood into the lungs occurs when the patient recovers, but blood cannot continue to occupy the larger tubes without producing suffocation.

The next problem to solve is the subsequent condition of the regurgitant blood, and first it is necessary to point out that no putrescence of the blood takes place in these cases. No evidence has been brought forward by Morton, Hoffmann, or Niemeyer to show that putrefactive changes do take place in the blood-effused into the blood under these conditions, and I have examined a very large number of cases without detecting the slightest odour about the blood thus inhaled remaining in the lungs sometimes for months after the date of the last blood-spitting, and expectoration of blood may extend over a long period without evidence of fœtor. It is quite true that if gangrene occurs the blood effused will share in the general process, and if blood be freely mixed with secretions exposed to air it may speedily become offensive. This is notably the case with blood effused from parts above the larynx, especially, for example, in epistaxis, but it is entirely contrary to my experience that clotted blood in the lungs should putrefy. The very occupation of the alveoli and the clotting in the bronchioles prevents any admixture with air and any special organism that might induce a septic condition.

In brown induration of the lungs, which is a very chronic process, blood is frequently extravasated, and the remains of pigment found in the alveolar epithelium, but there is never evidence of putrescence.

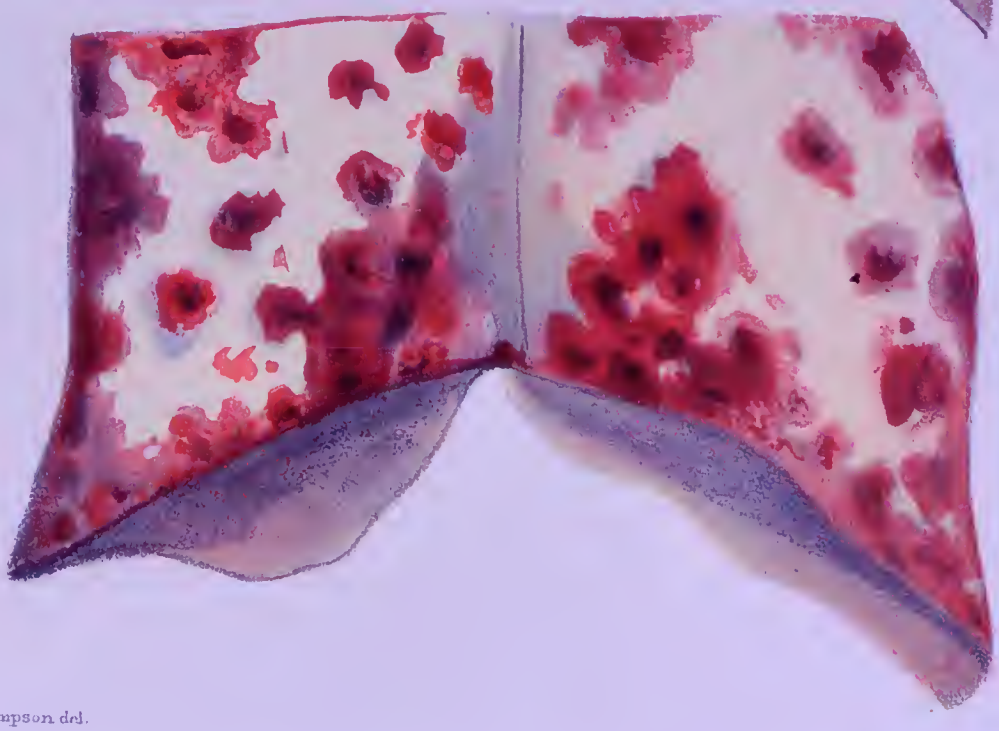
Embolism of the vessels of the lungs, leading to a wedge-shaped fibrinous mass formed from blood, is another instance to the contrary. Reasoning from such analogy as this condition exhibits, it might be assumed that blood would, in the process of time, lose colour, become coagulated, and at length contract into a hard white fibrinous mass.

This is notably the behaviour of blood not exposed to the air, and this is indeed exactly what takes place when blood is driven into the pulmonary tissue.

In a large majority of fatal cases which have come under

Fig. 1. Plate 1.

Fig. 2



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my observation at the Hospital for Consumption blood-spitting has occurred in varying quantities: and in a number of cases the relics of blood are to be found in the presence of hard fibrinous nodules, often deeply (though not always) pigmented, which are found usually in special habitats of the lung, which they assume by virtue of the peculiar influence of inspiration.

Examples must be given to show that this is the case, but it must be borne in mind that they are very frequently to be found if only the observer knows what to look for, and how to look for it. Having shown what are the appearances to be expected in a case of recent bleeding, I will quote a case in which early alterations were taking place in the blood, and by a sequence of cases the different stages in the process may be traced.

Case XV.—A man, aged 35, without any history of hereditary predisposition, had raised blood five weeks before admission into hospital. On two occasions half a pint had been ejected; from that time no recurrence of bleeding took place, but he developed symptoms of ascites which subsequently proved fatal.

P. M.—The liver was in a very advanced condition of amyloid.

The left lower lobe was found engorged with blood, evidently the result of inhalation, the tissue not being altered nor lacerated. A large patch of dark clotted blood occupied a portion of the lobe, the central parts of which were losing colour, and ovoid masses of granular texture, with little patches of dark pigment in the centre, were being formed. In these masses the openings of patent bronchioles were seen, and the pigment was being deposited in the lymphatic tissue surrounding them.

This condition had existed from three to four months, and the blood showed no signs of putridity.

As the process of absorption, decoloration, and fibrination goes on, the outlying portions of the blood disappear, while the central nodules become hard and white, and alone remain to show what has taken place.

In some examples these nodules may be found of different sizes, and varying in colour, some being of a light red colour, others of a lighter hue, and others again of an ivory white. They are generally mottled with old blood pigment, which collects, especially around the bronchioles, in small black

granules. In some cases all trace of pigment has been completely removed, and only a hard ivory-like fibrinous mass remains.

On the opposite page will be found illustrations of various alterations which inhaled blood undergoes.

Fig. 1 represents the condition of the base of the left lung in Case XV. It shows the clotting and loss of colour to which the blood is subject after the lapse of from three to four months (in this case).

Fig. 2 shows the condition of the base of left lung in Case XVI. The nodules had not altogether lost their colour, some were still blood red in the centre, less red at their circumference, others were of an ivory white ; all had pigmented bronchioles.

Figs. 3 and 4 are taken from Case XXIII. page 60. The first depicts the tendency to separation by traction from the rest of the pulmonary tissue, which is sometimes shown by some of these nodules. In this case softening was commencing at the upper part. Both processes are shown completed in fig. 4 from the same case. The capsule in this instance was completely separated and empty.

Case XVI.—Henry C——, aged 22, under the care of Dr. Powell. No family predisposition. He was first attacked with blood-spitting in 1874, and again in the month of June 1877, when he had copious bleeding; after that he had occasional attacks, once in November to the amount of 13 oz. He died on December 27, 1877.

P. M.—A large cavity, with obtruncated ends of vessels, two of which were plugged by reeent clots, occupied the upper lobe of the right lung.

A number of well-defined circumscribed oval nodules oecupied the middle portion of the lung, many of which were eroded and softened; pigmented bronchioles were found in the centres.

The lower border of the lung was firmly adherent to the diaphragm, and in the anterior inferior portion were found a number of similar rounded nodules, varying from $\frac{1}{8}$ to $\frac{3}{8}$ of an inch, with the openings of bronchioles in their centre, which were pigmented. The glands throughout were pigmented and of a dark black.

Left lung.—This was in a less advanced condition. The upper lobe was scooped out into small cavities about the size of a small filbert, which showed yellow matter close to the circumferenee.

In the middle axillary region were numerous hard nodules the

Fig. 1.

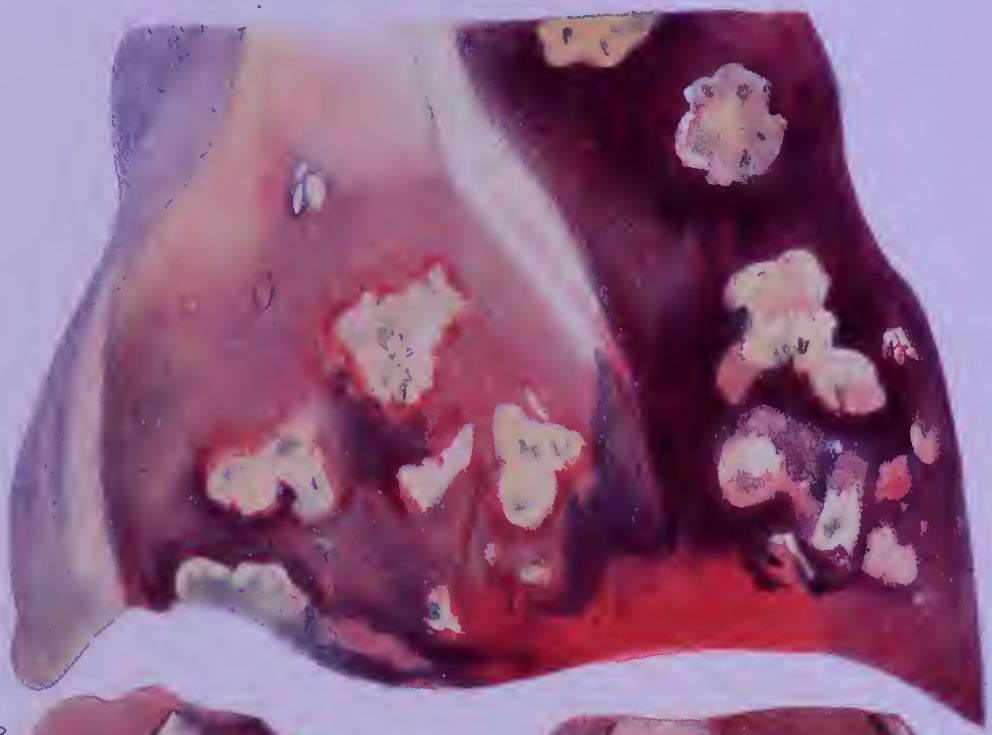

Fig. 3.

Fig. 4.

Fig. 2.

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Fig. 1

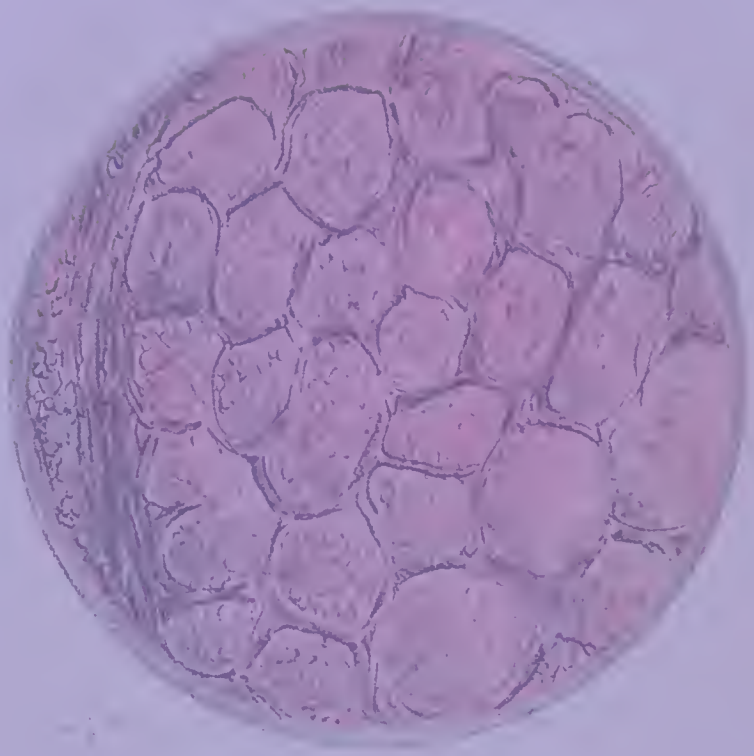
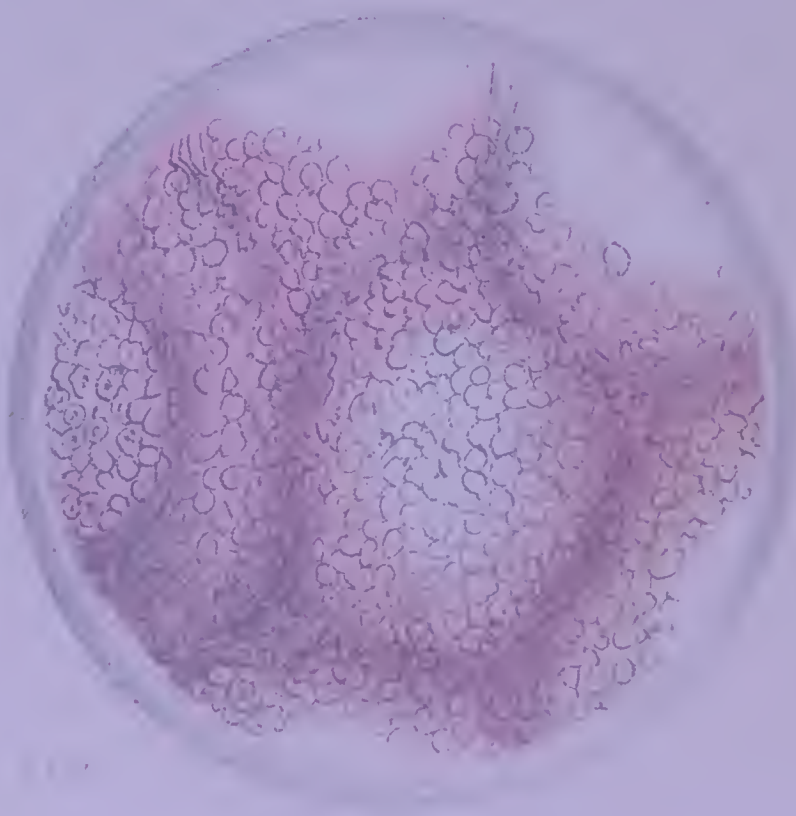


Fig. 2



size of a pea, close to the periphery. Also quite at the base were found a number of similar nodules. Some of them were apparently recent, the colouring of the blood being still visible.

Case XVII.—John S——, age 35, under Dr. Pollock.

Hereditary predisposition, *nil.*

Epistaxis in 1863 severely.

Hæmoptysis in 1875, and in January 1876—a pint, and half a pint, at the time; occasional hæmoptysis of less amount since, but not for three months before death.

P. M.—Right lung adherent at apex, puckered in front, the anterior border emphysematous. Base congested. The lower lobe contained some small solitary cavities at the upper part, and quite at the base, close to lower anterior margin, a few hard fibrinous nodules, the size of a pea.

Left lung.—Large cavity as big as an orange: at the lower portion, amidst intense congestion, nodules much pigmented in their centres, white in middle, shading off into red. Some ecchymoses on the surface of the pleura. Liver fatty. Kidneys granular.

When a recent specimen is examined from a lung of a patient who has died from hæmoptysis, the blood corpuscles are seen not only filling the lumen of the air sacs, but also apparently packed in the alveolar tissue, into which they have been driven by the acting force. This is represented in fig. 2, Plate III. which is taken from the same lung as fig. 1, but the latter represents the microscopical appearance of an old blood residue just described. Fig. 2 is evidently due to the condition of the last fatal hæmorrhage, the blood globules being well defined.

It is very probable that the thickening of tissue which is found in so many cases of old nodules is due simply to the infiltration of the pulmonary tissue with the blood cells, which lose colour, and thus leave a thickened white tissue. In some instances irritation seems to have been caused, and in this case this condition may have resulted from the contamination of the blood, during its passage into its resting place, with septic matter derived from some other part. This is an extremely difficult point to settle; but I cannot help thinking that pure blood can have little, if any, irritating effect on tissue, and if irritation is shown it must be due to some accidental quality of the blood, or some poisonous addition to it.

The microscopical appearance of these nodules consists of a group of alveoli, firmly packed with a semi-opaque homogeneous fibrinous material, and there is some thickening of the alveolar tissue, and also of the interlobular tissue, which thickened tissue forms the limiting capsule.

Although to the naked eye the vicinity of these nodules may indicate nothing special, yet, when examination is made with the microscope, the alveoli and bronchioles are found packed full of blood, the blood globules being more or less well defined according to the period at which blood has been inhaled. If careful search be made they will generally be found to be present where there is a previous history of copious hæmoptysis.

Their special habitats are the summit and middle part of the upper lobe, the middle axillary region, between the third and fifth rib, close to the pleura, the anterior inferior border, and the middle part of the base, corresponding to the summit of the arch of the diaphragm.

In women there are some peculiar characters which strengthen the argument that these nodules are due to inhaled blood. The nodules at the base are much smaller than they are in men, while the middle nodules are larger and more central. This is to be attributed to the sexual differences of respiration.

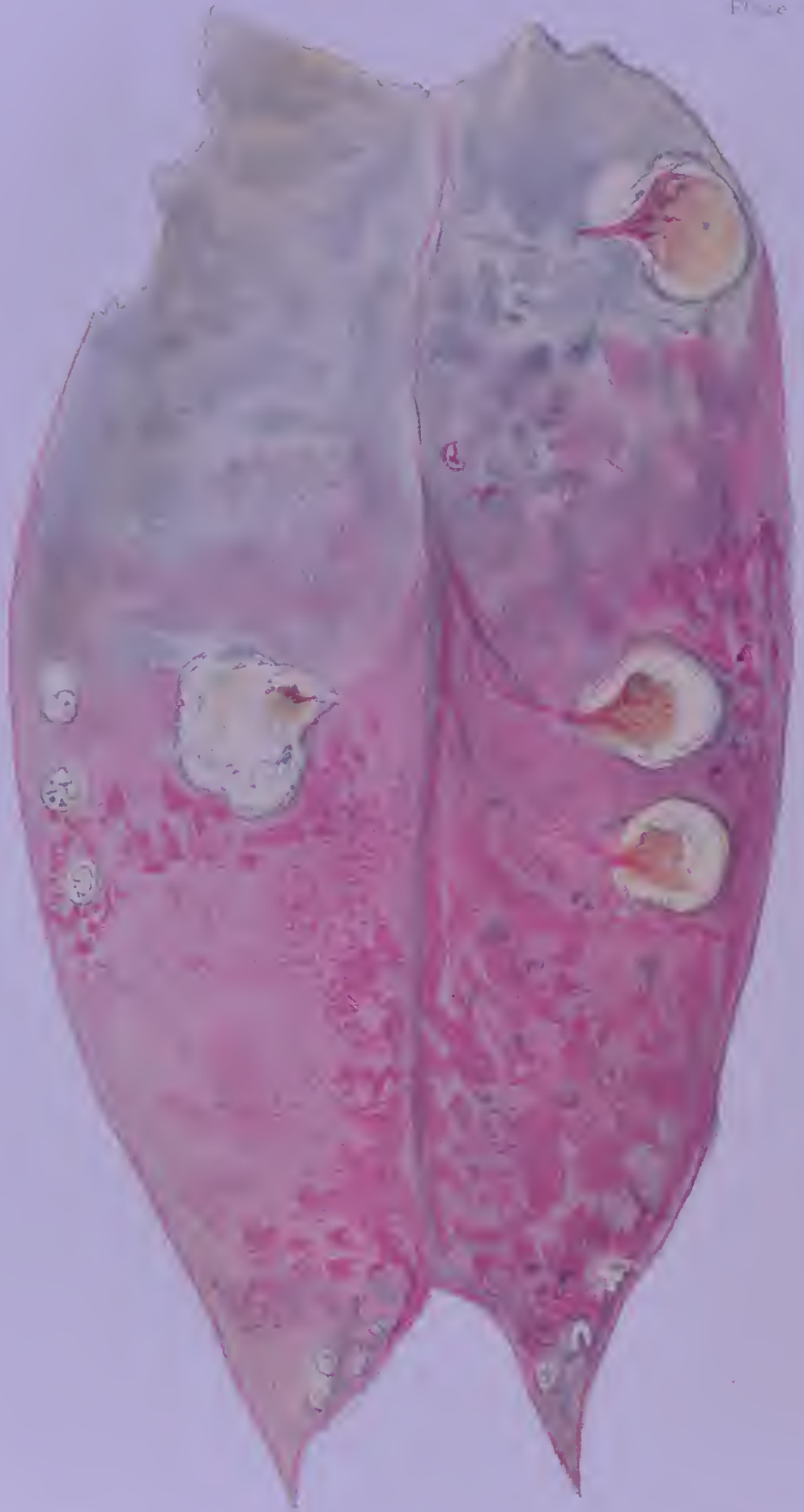
Case XVIII.—Mary S——, age 33, had suffered from cough for some time. Six months before death pulmonary bleeding occurred to the extent of a teacupful.

She subsequently died of hæmorrhage.

P. M.—Right lung was excavated by an irregular cavity situated in the upper lobe.

The middle lobe contained some hard large nodules, the size of a middle-finger nail. They were found below the main bronchus, and about an inch from the periphery. One of these nodules was softened, and from the upper part of the capsule was a small aneurysm the size of a millet-seed, which was the source of the fatal bleeding. The small cavity was filled with clot. Other smaller nodules were found close to the periphery in the middle axillary region.

The base was carefully examined: no nodules were found at the inferior posterior border, none in the middle of the lower surface, but a few small ones the size of a pea were found in the lower anterior border.



Left lung.—Contained a cavity with ends of vessels exposed, but not pervious. Small fibrinous nodules were found in the middle axillary portion.

Case XIX.—Mary Ann W——, age 30, under Dr. Pollock.

Blood-spitting once only, eleven months before death; it was then profuse. Death from phthisis.

P. M.—Right lung adherent. Upper lobe excavated into a large ragged cavity with vessels much exposed. No recent plugging of vessel; no aneurysm.

Left lung.—Distended and emphysematous.

Two nodules as large as haricot beans were found in the summit of the lung near the periphery. The lung surface above these nodules was puckered and retracted.

Two were also found of smaller size, as big as a pea, in the middle axillary border. None were found in the base.

Plate IV. is a drawing of the right lung in Case XVIII. It is a good example for showing the positions of the nodules, some of which, as it will be seen, have undergone softening, evidently from contamination with matter from the bronchial tubes. The upper portion gives a shadowy idea of the cavity, which was not more accurately expressed, inasmuch as it was intended only to indicate the positions and appearances of the nodules.

Attention must be directed to the peculiar isolated position of the nodules, especially at the lowest border.

To determine in a given specimen whether the irritation which is shown in the alveolar tissue is due to the presence of blood, to the addition of some septic poison, or to a previous condition of disease, is one fraught with the greatest difficulty.

Looking at the subject from general pathological knowledge and from the experiments already made on this subject, the conclusions which seem most accordant with the facts of the subject are as follows:

The invasion of a healthy lung by extravasated healthy blood is probably not followed by any irritation of tissue.

It is not so easy to determine this point as regards a lung in a condition of disease, and I can only say that, in this case there is considerable risk of irritation, which is probably proportionate to the activity of the disease in progress, and which may be due solely to the admixture of morbid secretions with the blood in its passage through the bronchial tubes.

Whether the blood of a syphilitic patient, for example, is capable of setting up a specific irritation of the pulmonary tissue, is a point I am unable to decide.

I have seen no instance to convince me that the extravasated blood of a tuberculous patient can cause tubercle, and I should be inclined to receive any evidence pointing to such a conclusion only after such evidence had been carefully investigated.

These observations are in accordance with the results of experiments that have been made on animals in Germany. Hertz discusses this point in Ziemssen's 'Cyclopædia,' and the passage may be quoted here (p. 306, vol. v.).

> Perl and Lipmann, experimenting upon rabbits and dogs, opened the trachea and injected into the lungs several grammes of blood drawn from the jugular vein.
>
> Examination at the end of twelve hours revealed the trachea, bronchi, and small branches of the latter perfectly free from coagula, while the bronchioles and alveoli were the seat of brownish red infiltrated foci, which by the third day had decreased in size, and by the end of the fourth week had entirely disappeared, without there having been at any time the slightest evidence of inflammation of the parenchyma resulting in cheesy degeneration.
>
> After the experiments by Sommerbrodt upon dogs, consisting in the injection of partly fluid, partly coagulated blood into the trachea, microscopical examination showed principally in the neighbourhood of the hilus, and more in the lower than in the upper lobes, and on the right side than on the left. Even at the end of three hours, and under abundant corpuscles, certain changes in the alveolar epithelium, consisting in an increase in size, cloudiness, and the like, which were most marked on the fifth day, and after that gradually diminished, so that in the fifth week every trace of these changes had disappeared.

It must be remarked that the artificial injection of blood is not identical with the insufflation from deep inspiration, although the above experiments serve to show that no putrescence nor caseous pneumonia results from the continued presence of blood in the lungs.

An indication of previous hæmorrhage, or of the presence of blood from some other condition, is obtained from the black

pigment, which is found almost always in the lungs of adults, and is due in great measure to the colouring matter of blood. In some instances the same appearance results from the inhalation of carbon, a fact which was long ago pointed out by English observers, and was until lately questioned by the Germans: the fact, however, has been established by some excellent experiments, especially those of Knauff; but this source of carbon must not be looked upon as the most frequent cause of the black pulmonary pigment. From the localisation of small particles in different regions of the lungs, and especially the base, which is the part most liable to congestion, and consequently to hæmorrhages, it appears to me to be certain that blood is the most ordinary cause of this pigment, and to it must be attributed local limited patches of the pigment.

Hence this black pigment, in the region of, or mingled with, diseased products, is evidence that blood has formed a portion of those products, or that a congestion has been caused by these products.

I am not aware that any distinction can be made by the microscope between the black of carbon and that of blood pigment, but in some examples blood crystals may be found which help to decide any doubt.

I have laid considerable stress on the presence of pigment in the nodules because it affords very strong evidence in favour of my position, but I by no means intend to assert that pigment is always present, or that a firm, hard, fibrinous nodule is not due to blood, if it has no pigment. I have seen many instances of such pigment-free fibrine, and in ordinary vascular clots, and even in bronchial plugs which may extend for some distance along a tube, no evidence of pigment can be obtained, and yet no one would hesitate as to the nature of such clots.

CHAPTER V.

SECONDARY CONDITIONS OF HÆMORRHAGIC RESIDUES.

The next point to determine is the alteration which blood extravasated into the lungs undergoes under certain circumstances, and to this we must look for the connecting link between blood-spitting and phthisis.

1. Pure blood ejected from the blood-vessels into the neighbouring tissue appears to undergo no further change than loss of colour and contraction. Small masses of semi-transparent pigmented fibrine, which can be attributed only to these extravasations, may be found unaltered. They may, however, share in a general destruction resulting from the development of a phthisical excavation or liquefaction, and the addition of old blood pigment to the secretion from a cavity gives a green tint to the yellow sputa.

2. Blood may, however, undergo an alteration probably from admixture with other matter, and may assume a calcareous degeneration which results in the presence of patches of calcareous granular matter mixed with pigment, and occasionally with the old colouring matter of blood.

The presence of this old yellow colouring matter, which I found in the case recorded below in large quantity, and the quantity of black pigment with which the inorganic and organic residues were mixed, afford conclusive evidence in favour of the previous presence of blood. I am convinced that such calcareous patches have often been wrongly attributed to tubercular conditions, and in proof of this I have lately put up in the museum two apex patches an inch long, one fibrinous and pigmented, the other calcareous, which were found in the right lung of an old man who died from advanced mitral disease, and who had suffered from hæmoptysis some time before death.

No tubercle was found in this case, and there was no reason for supposing that he had been subject to tubercle.

These calcareous patches may remain for a considerable period enclosed and imprisoned in their capsule, but they have a tendency, especially when situated in a very mobile portion of the lung, for example in the middle axillary region near the periphery, to rub an exit for themselves into the neighbouring bronchus simply by the constant attrition arising from a succession of expansions and compressions of the lung.

There is a specimen of this in the museum, in which a circular collection of calcareous matter much pigmented is forcing its way into a near bronchus which appears to have been previously occluded.

It is a matter of great difficulty to determine with certainty the exact previous condition when an empty cavity is presented to view, but those that result from the evacuation of these calcareous residues are marked out by a peculiar condition of capsule, which is uniformly thin, pigmented often, and generally retracted with the mark of contraction at the surface, which externally is puckered and knuckled in.

The following instance is apparently reliable, as there was only one circular cavity in a peculiar locality, the upper portions of the lung being occupied by calcareous patches which had not been removed.

In the left lung a circular, well-defined cavity, one inch in diameter, was found in the lower part of the upper lobe; the cavity was close to the periphery, was slightly retracted at this border, and pigmented. It was smooth-walled and non-secreting. Communication was established with a bronchial tube, a quarter of an inch in diameter, the place of juncture being marked by a distinct narrow contraction of the tube before passing into the cavity. Calcareous patches with similar capsulating membrane and pigmented were found in the upper part of the same lobe.

These cases are not common, but we have thus a connection established between blood residues and the formation of cavity.

In the following plate (Plate V.) I have given instances from an example which I have already partially described in Case XV.

The conditions are so important that it is necessary to repeat the case in full with all its details.

Case XX.—John W——, aged 35, hospital porter, admitted October 9, under Dr. Pollock.

Hereditary predisposition, *nil.*

Personal history.—Rheumatic fever in 1870. For four months before admission had suffered from cough with hæmoptysis. Hæmorrhage of black blood on two occasions to the amount of half a pint, the last time five weeks before entering the hospital. Considerable loss of weight. Had suffered from diarrhœa.

On November 20 he began to suffer from ascites, and the urine was found to contain a large quantity of albumen, sp. gr. 1015, 50 ounces in quantity.

He continued to suffer from diarrhœa, and occasionally vomiting, during the rest of his lifetime, but there was no return of hæmoptysis during the time he was under observation. He died on December 28.

Synopsis.—No hereditary predisposition. Hæmoptysis to the amount of half a pint twice; the last time being nearly four months before death.

Sectio cadaveris.—December 22.—A small depressed cicatrix of old sore on the penis.

Right lung very adherent, adhesions universal and thick. The pulmonary tissue was generally very much congested, soft, and dark, containing a considerable quantity of blood. In the middle of the upper lobe and middle lobe were found two irregular patches of mortar-like matter, and surrounded by thick, black, pigmented tissue; the lower patch having a number of small, rounded, mortar-like masses, the size of mustard-seed, outlying and similarly surronnded by thickened pigmented tissue. No tubercle was detected.

Left lung.—Adherent behind, with long fibrinous bands of attachment in front. Three irregular patches of mortar-like matter, similar to those in the right lnng, were found occupying the lower and posterior portion of the upper lobe. A circular, well-defined cavity, one inch in diameter, was found in the upper lobe. This cavity was close to the periphery, and was pigmented at its margin; the lining membrane had here contracted and become depressed; it communicated with a bronchial tube of some size (quarter of an inch). It was evidently the result of a complete clearing ont of a mortar patch similar to those already described. These mortar

Fig. 2.

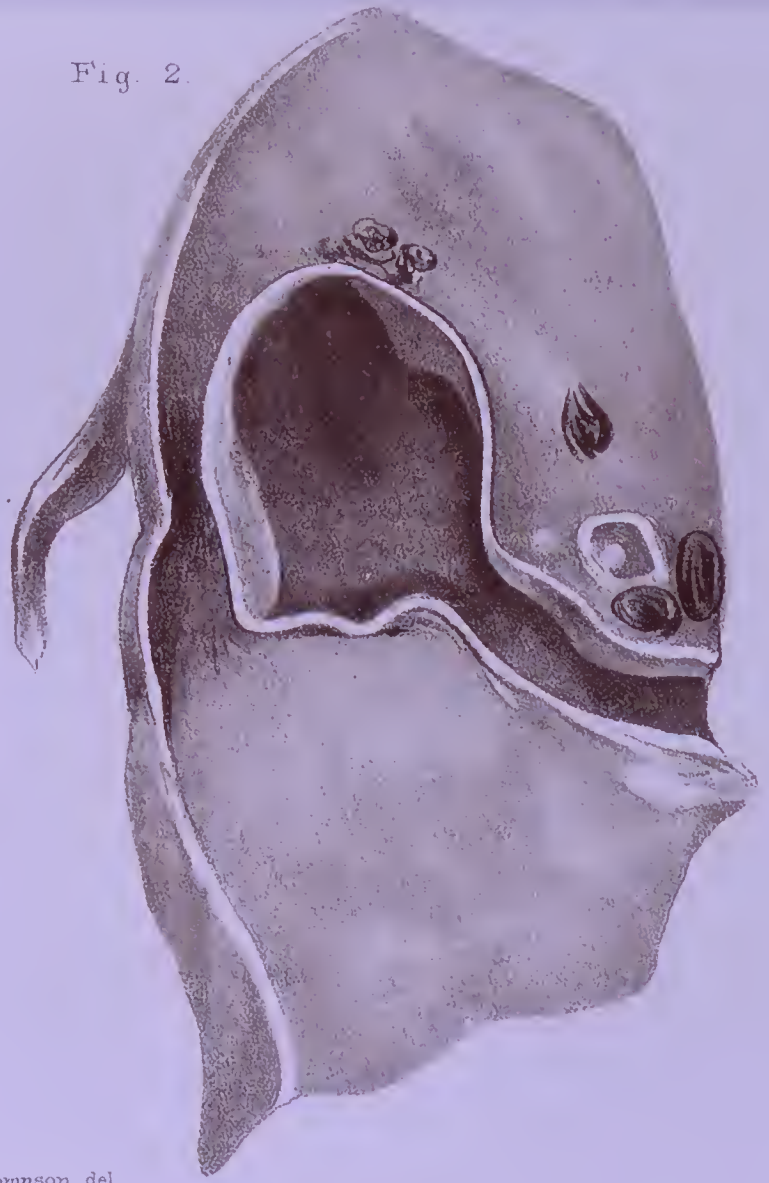


Fig. 1

R.E Thompson del

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patches were found to contain small masses of bright yellow pigment, evidently colouring matter from old blood. The lower part of the lower lobe was found full of semi-decoloured blood surrounded with blood-stain and congestion.

Liver was covered with recent lymph, and was found in a very advanced amyloid condition. The peritonæum was also covered with thick fibrinous lymph, and the kidneys and intestines were found to be amyloid. The large intestine was studded with a number of old thickened ulcers, which were probably of dysenteric origin.

3. The fibrinous nodule which is found very frequently, undergoes changes of a peculiar kind, and results in the formation of cavities, which lose their specific character in the general destruction which follows the establishment of a condition of phthisis, but I am fully persuaded that they form the nucleus of initial softening far more often than is at present suspected. My reason for this opinion is that I have seen so many cases in which multilocular cavities of the upper lobe constitute the main condition of a phthisical lung; and in many of these cavities remains of these fibrinous nodules may be seen, either clinging in a remarkably persistent manner to the walls of the cavity, or sufficiently lining the walls to give an indication of the original condition of the cavity; these fibrinous residues must, however, not be confounded with caseous remains, to which they have a close resemblance.

These nodules may result in the following conditions:

1st. Softening may occur around the periphery, or in the centre, and lead to general liquefaction of the nodule.

2nd. They may separate from the surrounding tissue by traction.

1st. When the nodules soften, as a general result of pulmonary softening attacking the upper lobe, they sometimes soften at the circumference, and portions are found still adherent at parts, but generally separating from the interlobular capsule.

If they soften and the matter is not ejected and cleared out, they result in the formation of a cavity filled with a glairy fluid of a yellow colour, quite peculiar in character, which might be

mistaken for pus, but under the microscope the appearance of irregular *débris* only is discovered; this no doubt constitutes the cavity which has been so well described by Morton in one of his cases of Phthisis ab Hæmoptoe, and to which he gives the descriptive epithet 'mellea,' the contents of which certainly closely resemble Narbonne honey. For a long time I was quite at a loss to account for these peculiar cavities to which my attention had been directed for some time, and it was only from the detection of the nodules in the process of softening that I was able to come to a conclusion on the point.

When these cavities are cleared out of their contents the walls are seen formed of thickened tissue, and often remains of the fibrinous matter may be found, on close inspection, clinging to the walls.

The position and peculiar isolation of these cavities also help to their diagnosis. Students, as a rule, are at the present day too apt to pass over all pathological conditions to which the label phthisis can be attached as uniform and uninteresting, and the consequence is that the whole subject has been treated with undeserved neglect; nothing in the whole range of pathology is more difficult, and in my opinion nothing more interesting, than to endeavour to establish the previous history of a phthisis by the indications afforded after the death of the patient, but the greatest caution and discrimination is requisite to avoid falling into errors.

Case XXI.—Charles B——, admitted October 18, 1877, under Dr. Powell. Began to suffer from hæmoptysis in 1864; again attacked with very copious hæmoptysis in 1873. This returned five months before admission, but it did not recur again. No hereditary predisposition,

Sectio cadaveris.—Left lung large; the upper lobe contained two cavities as big as a walnut, $1\frac{1}{2}$ inch across, filled with glairy yellow puriform fluid; the tissues surrounding these cavities were deeply pigmented and thickened. In the neighbourhood at the back were found some smaller well-defined oval cavities filled with the same yellow matter.

In parts some of the small terminal bronchioles and alveoli were found choked with white fibrinous material, the residues of blood.

Much the same appearance was found in the right lung, only it was less affected.

Case XXII.—Elizabeth L——, aged 30, under the care of Dr. Pollock. No hereditary predisposition.

Hæmoptysis profuse four years ago—streaks since at times.

Right lung.—Upper lobe in a condition of pigmented thickening of a specific character; excavated at back by a cavity the size of a Maltese orange.

Several pigmented nodules, some as large as a pea, others larger, were found in the middle axillary portion, and a few in the anterior lower border.

Left lung.—Upper lobe congested and previously thickened. At upper axillary portion of lower lobe a cavity was found, the size of a filbert, quite empty, deeply pigmented, with cicatricial marks at the periphery, where there was also depression. On close examination portions of fibrinous nodule still remained in the walls not liquefied.

These nodules may undergo partial softening, that is to say, portions may be eroded by admixture with matter from a distant source. In this case the nodules are found eaten out bit by bit, that portion being first removed which is nearest to the bronchiole, and occasionally the nodules are found stained at their parts with sanious matter derived from a superior cavity; or two nodules may be found, one above the other, and the lower one will be softened while the upper one remains intact; all these instances are but examples of the inspiratory force as an agent of disease.

This last condition never occurs without the previous existence of a secreting cavity, and it is set up therefore by phthisis, and is a secondary result of a cavity.

2. The hard, unyielding nodules, firmly held by their enveloping capsule, have a tendency to tear away simply by traction from the lung tissue in the neighbourhood, and this especially happens if there is any tendency to softness from a condition of congestion.

The nodule then tears away, and may give rise to hæmorrhage by the laceration of surrounding vessels.

Sometimes the separation of the nodule is so complete that the capsule is left loose in a cavity of its own formation. Twice I have seen this rare condition of which the details are given in the following cases:

Case XXIII.—H. G. C——, aged 22. Admitted under the care of Dr. Pollock, May 8, 1877.

This man had been subject to hæmoptysis since 1874, sometimes copious.

Sectio cadaveris.—Right lung was excavated at the upper lobe: a large sanious cavity occupying the whole of this portion.

In the lower border was found a cavity as big as a walnut, which contained, lying free within it, a capsular bag an inch long, filled with recently clotted blood, the remains of a recent hæmoptysis, which could be shelled out of it, and was evidently of much later date than the rest of the capsule. On making sections of this capsule it was found formed of interlobular tissue much thickened, and under the sac were found remains of alveoli packed with fibrine, but not so closely packed as entirely to compress the alveolar vessels.

Case XXIV.—John M——, aged 34, was under my care for some years with hæmoptysis, which began first in 1873. In 1875 he had a very severe attack, and lost more than a pint of blood.

In 1877 he lost two pints of blood, and finally at the end of the year he was suddenly seized with profuse hæmorrhage, which proved fatal.

Sectio cadaveris.—Left lung very much retracted and dwindled. It contained a ragged aneurysmal sac, which had burst, and near this were found two or three curdy masses, three-fourths of an inch in diameter, which were softened and eroded at the upper portions and below were undercut, as it were, separated from the surrounding pulmonary tissue.

In the right lung, under the fourth intercostal space, in the anterior axillary line, a cavity was found, on cutting into which there appeared a round yellow bag or capsule lying loose and empty in the cavity.

There was no blood nor purulent secretion in the cavity. The sac would have fitted the tip of the little finger, was rough and ragged inside and out; it was evidently the remains of a curdy mass, similar to those in the other lung, which had softened, emptied itself, and separated from the surrounding lung.—(See Plate II., figs. 3, 4, p. 48.)

In one case reported by Dr. Goodhart to the Pathological Society, fatal hæmorrhage resulted from the tearing of vessels owing to the traction of one of the nodules.

Finally, with regard to the ultimate condition of these

hæmorrhagic residues, it is necessary to consider the possibility of tubercle as a result of infection with their softened products.

As yet I have seen no case which lends colour to the theory propounded by Andral, that hæmorrhage can directly (by infection with blood from an individual predisposed to tubercle), lead to the formation of tubercle by the irritation set up by the blood itself.

Were this possible we should, I imagine, often see tubercles situated around the hæmorrhagic nodules, but this I have failed to establish in any of my numerous cases. The case reported by Andral on which he relies for a support of this theory, shows the very great care that is required in coming to conclusions about the matter; and I must point out that the dates of hæmorrhage must be in all cases carefully examined, and the corresponding conditions of blood be noted, as well as the probable dates of the deposit of tubercle. My experience as regards the formation of tubercle is that the tissue required for its growth varies in great measure according to the age of the individual: in childhood it requires only days, in advanced age it requires months for its development; and the mere presence of unaltered blood in a region affected with tubercle would lead me to conclude not that the tubercle is the result of the hæmorrhage, nor indeed the cause of it, but that they are both, blood and tubercle, landed in the same locality at different periods by the agency.

Again, I have seen old white nodules, which were certainly of many months', and probably some years', standing, in the same locality with a recently formed glistening young tubercle, but experience, as regards the time necessary for the production of the separate states of the blood product and the tubercle, compels me to separate the two as having no interdependence. This is an extremely important point, and must be steadily borne in mind in all future observations.

But the liquefaction of the nodules, and their ejection along the bronchial tubes, renders the risk great of some portions being returned by the inspiratory act into some other portion of the lungs, and thus by a repeated act of infection lead to the formation of tubercle.

Under these circumstances it seems evident that some new

septic condition has resulted, which may induce a special property of infecting.

The greatest caution should be observed in coming to any conclusion from the presence of tubercle in regions occupied in part by traces of old bleedings, and the kind of evidence that should be looked for appears to me this: the presence of grey tubercle in the vicinity of a nodule of blood-stained fibrine, tubercle requiring in the adult from six weeks to three months for its formation, while the colour of blood requires about that period before any change is effected.

It is a little difficult to find convincing cases for record in which all the cavities can be traced distinctly and solely to the previous presence of hæmorrhagic residues, and in all cases where the excavation is the result of previous inflammatory disease no deductions can be made as to the original source of the infective matter necessary for the production of tubercle.

In the case already given at page 58 (C. B., Case XXI.) no such complication was present, and the presence of tubercle was probably due to the infection with the liquefied products of the blood residues: the amount of tubercle was not great, and this supports the conjecture.

In this case the bleeding was originally the result of pulmonary apoplexy (to judge at least from the pathological condition of the lung) dependent upon alcoholism: the liver being found after death in an advanced state of cirrhosis. There was no hereditary predisposition in the case, and there was no evidence, clinical or pathological, that the patient was the subject of phthisis in the general acceptation of the term.

The only cavities found were the result of the liquefaction of the fibrinous nodules, but at the middle part of the lower lobe of the right lung a few racemose groups of recent tubercle were found.

It is necessary to caution the inexperienced observer against the appearances sometimes resulting from the presence of small nodules of fibrine, no larger than a pin's head, in the air sacs. These little nodules are harder and more opaque than tubercle; the microscope at once reveals their nature, and they must not be mistaken for tubercle.

CHAPTER VI.

THE INHERITED PREDISPOSITION TO HÆMORRHAGE.

The manifestations of disease which are the result of inherited proclivities, whatever be the nature of the inheritance, are often differently localised in members of the same family, and in no cases are more diversities exhibited than among the offspring of phthisical parents.

In the manifestation of inherited gout those parts are most likely to be affected which from use receive the greatest localised supply of blood in comparison with parts less exercised, and this result is accounted for by the theory that there is an inherited tendency to the formation of a special chemical substance which accumulates in the blood unless special means be taken to effect its removal, and those parts are likely to be attacked to which by use a large supply of blood determines, the special poison being present in the blood.

Such a reason will not satisfy the various phenomena of inherited phthisis, and consequently some other cause must be sought for the determination of this disease to special localities.

But phthisis must not be looked upon as a simple and uniform disease like gout; its varieties are so protean in form, that to consider merely whether a predecessor has had phthisis, without endeavouring to investigate the special features which it has assumed is not sufficient for the purpose in view.

The inherited predisposition to disease which is engendered in the offspring of a phthisical parent is a most complex heirloom which may be handed down as a whole or only in parts; like the Roman lictors' fasces, it may be held in a bundle by one individual, or split up into parts and distributed among many. The comprehensive disease which is known under the name of 'phthisis' may start from various points, and the dif-

ferent roads by which it travels may converge towards one and the same terminus, and phthisis must be looked upon as the ultimate result of many different forms of disease rather than as the *fons et origo mali* from which the initial diseases flow.

By this way of looking at the subject we put out of view the theory of Laennec, which I hold, with many others, to be untenable, and instead of attributing all those forms of disease which may eventually terminate in phthisis to an originating stage of tubercle, I must express my conviction that the nodular tubercle is the result of a secondary process, and that phthisis, whatever be its origin, ultimately tends to its formation.

This opinion does not interfere with the idea that some forms of phthisis are especially tubercular in character from a very early stage, it applies solely to the nodular form of tubercle, which must not be considered identical with a general tubercular condition, although the nature of the process may be similar; but as my arguments are little concerned with this point, I need not enlarge upon it in this place.

The two characteristic attributes which signalise the inherited predisposition to disease derived from a phthisical progenitor are especially shown in the manner in which such a tainted constitution deals with inflammation and irritation. The physician is always suspicious of such a predisposition when he finds his patient very apt to fall ill from trivial causes, very slow to recover even from the most trivial forms of disease.

The two features which mark the phthisical constitution are:

1. Susceptibility to external influences.
2. Indolence in the repair of structural destruction.

The first attribute, susceptibility, is not only constitutional and general, but it is often specially localised.

The constitutional susceptibility is shown in the exaggerated influence of extraneous causes, shock, cold, &c. as compared with what occurs in individuals free from the inherited taint. The local susceptibility is shown in the liability to irritation to which certain tissues and structures are especially prone, a determination of disease to certain localities which is sometimes different in members of the same family.

Indolence in the repair of structural destruction is dependent

not only upon a constitutional feebleness which affects the growth of tissue, but also upon an indolence in the removal of morbid products which appears to be the result of a delicate condition of the lymphatic system.

Both of these characteristics are explained by the transmission of structural peculiarities, which we know, from innumerable instances of other kinds, in animals as well as men, is exactly that kind of inheritance which passes from parent to offspring, and there is no violation of the ordinary laws of descent in supposing such transmission to take place.

There is no difficulty with this theory in view (and I would say it is identical with that which has been most ably urged by my friend and colleague Dr. T. H. Green in his 'Treatise on the Pathology of Consumption') in ascribing non-inherited pulmonary phthisis to causes of a similar kind acquired by individual conditions and not by inheritance.

In the case of inherited phthisis we have a congenital delicacy of structure, while in the case of non-inherited phthisis the delicacy of structure is due to some incidental condition affecting the constitutional growth of the individual, and preventing the healthy renewal of tissue as it yielded to the wear and tear of ordinary life.

On the contrary, the theory of inherited tubercle as a special entity affords no help in bridging over the connection between inherited and acquired phthisis; nor does such a theory account for the various forms of disease which initiate phthisis and form part and parcel of the phthisical heirloom.

With the theory of structural imperfection, the subsequent period of renewed growth and vitality can be credited on a perfectly intelligible hypothesis with the obliteration of the scrofulous tendency, the large cell formation from peculiar structure, which passes away in adult life as the tissues become renewed under more vigorous conditions; if such revitalisation does not take place, scrofula developes into pulmonary phthisis, which still retains the peculiar tendency to exhibit large cell growths; and this theory also accounts for the peculiar relation which forms a link between hæmorrhage and phthisis, and which I shall proceed to illustrate.

The theory of a special fragility of vessels which was put

forward by Niemeyer has met with most undeserved neglect, and has been hastily put aside without reason, and although this theory was not developed, and was not satisfactorily worked out, the tendency to bleeding which is exhibited in many families, sometimes combined with, sometimes distinct from pulmonary disease and phthisis, a tendency which is also distinctly transmitted by inheritance, points not only to a relationship between hæmoptysis and phthisis, but to hæmoptysis and hæmophilia.

The general tendency to structural delicacy exhibited in the offspring of a phthisical parent is perceptible even to the eye of the uneducated, and popular opinion as to the correlation of the two conditions suffices to establish this point; every one recognises the characteristic delicacy of appearance, features, skin, and general condition which as a rule marks out these individuals. Instances of every-day occurrence could be given to show that this general delicacy is accompanied by some local weakness, which tends to determine the effects of cold and the like to that particular spot, different for different members of the same family.

For example, the offspring of a phthisical parent may manifest a tendency to scrofulous disease—one in the glands chiefly, another in the joints, another in the skin—and it seems probable that the general constitutional delicacy may be accompanied in each case by a structural imperfection or non-development, an arrest of growth in some local part; it would also appear probable that the special local weakness may by inheritance be continued in the progeny of the so-affected individual. Hence I am inclined to believe that not only is a general constitutional weakness transmitted, but also local imperfections affecting certain organs may also be transmitted, and thus some reason is given for the special features which sometimes characterise the form of disease assumed generally by members of the same family.

This is the case in a remarkable manner with asthma, which is usually the result of a predisposition inherited from a phthisical parent or ancestor; it very rarely, except in gouty cases, occurs as a condition independent of such a predisposition. As a rule it does not terminate in phthisis, nor is it generally accompanied by that constitutional delicacy which belongs to

phthisis, it is often found among those who would pass muster with the healthy and robust. But in families of which one member is asthmatical, there is often a peculiar tendency to bronchial affections chiefly. It certainly appears as if the asthmatical disposition is something over and above the simple disposition to phthisis, and must be considered as separable from and not included in the general constitutional weakness which forms a main part of the predisposition to phthisis.

Following a similar line of argument, I conclude that the hæmorrhagic tendency which is found as a peculiar feature affecting many members of the same family, and may be exhibited in various ways with or without phthisis, is really a condition which may be specially transmitted, but is separable from and not necessarily included in that general structural delicacy which forms the main part of the predisposition to phthisis. Just as it is possible to encourage certain structural peculiarities by the selection which arises from inheritance, so it is possible that certain imperfections should be eradicated; and hence I believe that many cases of pulmonary hæmorrhage occurring from trivial causes without evidence, present or consequent, of pulmonary disease, are really due to a special predisposition inherited from, it may be, a far-off phthisical ancestor, the liability to phthisis having been in great measure eradicated while the hæmorrhagic tendency has survived. The two conditions must be considered separately, and it is upon the evidence of history, obtained in every case by a careful investigation of the characteristic features of disease as affecting collateral branches of the family as well as the immediate relations, that the diagnosis and prognosis of each separate case in great measure depends.

I will now proceed to relate a few cases out of many to show the correlation of the two conditions, and I would remark that it is a matter of no little difficulty to obtain comprehensive family histories—the knowledge of the children seldom extending beyond their own lives; hence it has happened to me occasionally to fail at a first inquiry in establishing a connection between the hæmorrhagic patient and the phthisical progenitor—the intermediate relations having evinced no special tendency to disease; but the history has generally been rendered complete, and the

disease traced back perhaps to a great-grandparent. The more experience I receive with regard to these cases the more probable this theory becomes, and the more separable the two conditions appear to be, so that, happily for the patients, I can with truth give a far more hopeful verdict on the case than I could possibly have given before these investigations were made.

The evidence of women regarding the condition and diseases of their parents and ancestors is far more complete than that of men, and to this partly must be attributed the number of women whose cases are here recorded, most of the men examined on points of history having shown a remarkable ignorance of any details respecting even the near relatives of their parents.

Case XXV.

Maternal great-grandmother died of phthisis at 29.

Her children were:

- A daughter who died of phthisis in youth.
- A daughter who suffered from severe epistaxis; no cough.
- A son who also suffered from severe epistaxis; no cough.
  - His eldest son always suffered from profuse epistaxis, brought on by any mental emotion; no phthisis.
- A daughter (*grandmother*), thought to be delicate in early life, but without pulmonary symptoms. Used to suffer profuse epistaxis, even after 70 years of age.

The family of the grandmother was:

- A daughter who died of phthisis at 25. She had hæmoptysis. No epistaxis.
- A son who suffered from congenital morbus cæruleus, and died at 17 of profuse hæmoptysis; without phthisis.
- A son who suffers from eczema, but is in good health.
- A son who suffers from bronchitis.
- A daughter (*mother*) who suffers even now, at 70 years of age, with epistaxis and eczema; no phthisis.

The mother's children were:

- A daughter who died of pulmonary disease, without hæmorrhage.
- A son who suffered from profuse epistaxis from 7 years of age, and died of phthisis with copious hæmoptysis.
- A son who suffered from epistaxis, profuse hæmoptysis, and pulmonary phthisis. He died from accident.
- A daughter who suffered from epistaxis and profuse hæmoptysis, and died of phthisis.

Two daughters who have suffered from epistaxis and hæmoptysis, without symptoms of phthisis.

A son who suffers from hæmorrhage from the bowels, which last symptom was also shown by all the family.

CASE XXVI.—Charles W——, age 25.

Father's father had phthisis. Father and mother healthy.

Father's brother died of phthisis.

Nine in the family—four brothers, five sisters.

Patient used to suffer from profuse epistaxis up to 20 years of age. Gums bleed on brushing, and are spongy.

Purpuric condition of cutaneous vessels.

Has had blood-spitting for four months.

Physical signs.—Moist crackle at both apices.

Sister also under my care, age 20.

Catamenia at 12; used to suffer from epistaxis.

Blood-spitting profuse two years ago.

She was examined by me first in 1875, and I then found some moist sounds at the right apex, but these dried up, and on examining her on December 24, 1878, I found no symptoms of pulmonary disease.

CASE XXVII.—Albert A——, age 31.

Father broke a blood-vessel three years before death, which eventually occurred from phthisis.

He brought up at first a pint of blood.

Family consists of four sons, two daughters.

Two sons and two daughters healthy.

One son died of phthisis.

Patient, after a trivial exertion, coughed up half an ounce of blood. Has had four attacks in four months; the last time, after walking home in the evening, he coughed up one pint of blood at midnight.

Has had epistaxis lately.

Has passed blood from the bowels.

Physical signs.—Dull percussion with viscid click under the right clavicle.

CASE XXVIII.—Isaac M——, 18.

Father had cough.

Father's sister died of phthisis.

Her daughter under my care with blood-spitting.

Father's family consisted of:

A daughter who died of phthisis and blood-spitting.
A son who died of phthisis and blood-spitting.
A daughter who died of phthisis.
A son, the patient.
A son accustomed to profuse epistaxis, now 20 years of age, without blood-spitting or cough.

Patient has never suffered from epistaxis.

Twelve months before visit, he was attacked suddenly on a Sunday evening with copious bleeding.

The blood recurred in July 1878, and again seven days before visit.

Case XXIX.—Walter J.——, age 20.

Father died of phthisis with blood-spitting.

Seven brothers and sisters in health.

Patient has had attack of blood-spitting three times.

On examination, slight moist sounds at right apex.

Case XXX.—William B——, age 38.

Mother subject to blood-spitting and doubtful phthisis.

One sister died of phthisis and blood-spitting.

Patient spat blood four years ago.

Has now a slight cough.

Used to suffer profuse epistaxis three and four times a week when a boy.

Physical signs.—Slight and scattered subcrepitant *râle*, right lung.

Case XXXI.—William H——, age 15.

Father's mother was phthisical, and died of blood-spitting.

Father's sister suffers from phthisis and blood-spitting.

Fourteen in the family, eight of whom have died.

Patient has had blood-spitting; no epistaxis.

Physical signs.—Crepitation of phthisis in both lungs.

Case XXXII.—Florence B——, age 15.

First consulted me in November 1877.

Father suffered from profuse blood-spitting and epistaxis; suffers from phthisis—age 43.

Mother died of cancer.

A sister died of phthisis at 19. She had profuse blood-spitting.

A sister suffers from cough and profuse epistaxis.

A brother has cough and epistaxis.

Patient had epistaxis in 1877, and blood-spitting.

Catamenia regular. No physical signs.

She consulted me again January 21, 1879, for cough and epistaxis. No physical signs.

CASE XXXIII.—Ann C——, 26.

Mother has cough, and suffers from blood-spitting.

Two brothers healthy.

Suffers from epistaxis.

Was confined three months ago, and suffered from flooding.

Catamenia very profuse two weeks ago.

Has had a cough.

Physical signs.—Harsh respiratory murmur, right apex.

CASE XXXIV.—Lucy W——, 20.

Father died of phthisis.

Ten in the family.

A brother with rheumatism. No bleeding.

A sister, profuse blood-spitting, died of phthisis at 18.

A sister, profuse blood-spitting, died of phthisis at 28.

A brother, profuse blood-spitting, died of phthisis at 22.

A brother died in the hospital with blood-spitting and phthisis.

A sister
A brother } quite well.
A sister

Patient suffered first from blood-spitting seven years ago.

Blood-spitting returned in September 1878.

Losing flesh, large pupils, thin skin, small chest.

Physical signs.—General crepitation over right lung, with pain on inspiration below clavicle.

CASE XXXV.—Sarah S——, age 40.

Mother died of phthisis.

Eldest sister died of phthisis and had copious bleedings.

Another sister used to have bleedings; is now 50 years of age.

Eldest brother used to spit blood, but is now well.

Patient used to suffer from profuse epistaxis between 12 and 13, and was obliged to have her nose plugged.

Spat a pint and a half of blood nine months before visit.

When examined the only indications of disease were some bronchial *râles*.

The following month there was a recurrence of bleeding to the extent of a quarter of a pint.

Case XXXVI.—Mary A——, 10.

Two weeks ago spat up one ounce of blood, which recurred three times. Spongy gums. A little crepitation in both lungs.

Father accustomed to have epistaxis.

Case XXXVII.—Arthur F——, age 4 years.

Family history.—Great-grandmother (maternal side) died of phthisis. She spat blood profusely.

Grandfather (same side) died of phthisis and copious blood-spitting.

The brothers and sisters (fifteen in number) of the grandfather died from the same conditions, out of a family of eighteen.

Mother has spat blood; had much flooding during the last two confinements.

Mother's brother suffers from blood-spitting and cough.

Patient has suffered from blood-spitting for six months. Has had epistaxis and bleeding from the gums. A small scratch on his arm from a cut with a ginger-beer bottle is reported to have bled profusely, saturating the bandage. Gums vascular and bleeding. The bleeding from the lungs, which is slight in quantity, has come on after a fit of passion. Has scrofulous glands at the back of the neck, for which no cause of irritation was found in the head or elsewhere.

Both mother and child were carefully examined, but I failed to find any physical signs, except a little harshness of respiration. The mother was a stout, well-made, healthy woman.

The child was well-made, but with a thin skin. There was no evidence of general hæmophilia as regards the skin.

Case XXXVIII.—Eliz. H——, age 19.

Maternal great-grandfather died of phthisis with copious hæmoptysis.

Both grandparents alive and well, without symptoms of disease; had the following family:

A daughter who died of phthisis with copious hæmoptysis. Catamenia always profuse.

A daughter who has symptoms of phthisis, and has had a family of three children, all of whom died of phthisis with hæmoptysis.

A daughter (mother) who has suffered from epistaxis, which is occasionally profuse—now at the age of 40. Catamenia occurred at 14, and always profuse.

Her daughter, age 19, has the following history: no cough; no epistaxis, but catamenia always profuse. Large glands; incipient ulceration of the bowels. Symptoms of phthisis.

CASE XXXIX.—Sarah M——, age 46.

Her father died of phthisis with copious hæmoptysis.

A brother died of phthisis, with copious hæmoptysis and epistaxis.

A sister suffered from profuse epistaxis and hæmoptysis; died of phthisis.

A sister suffered from epistaxis and hæmoptysis with phthisis: died from some other cause.

A sister delicate, with slight hæmoptysis.

The patient has been under my care for the last year. No epistaxis, but frequent profuse hæmoptysis, without evidence of phthisis.

CASE XL.—Louisa B——, age 10.

Maternal grandfather and five aunts on the same side died from phthisis, accompanied with severe hæmoptysis.

Mother, aged 30, had epistaxis when young, and her nose would bleed severely from the most trivial shocks. At 28 years of age she spat up more than a quart of blood without previous cough. She was delicate, thin-skinned, and small-chested, but showed no signs of phthisis.

The patient had not suffered from epistaxis, but the catamenia had first appeared at the age of 7 years, and she had menstruated regularly every six months since that time. She spat blood first in January 1878, a year before I saw her, to the extent of a pint, and this recurred between the catamenial periods. A thin delicate child, with bluish tinge and small chest. No phthisis.

These cases might be multiplied, but the evidence as it stands is sufficient to show that some families are especially affected with a tendency to hæmorrhage, whether pulmonary disease be developed or not, and it can no more be argued that in these cases the hæmorrhage from the lungs is the result of the presence of tubercle in the lungs than that the epistaxis is due to the presence of tubercle in the nose; the only conclusion is, that there is in some families a special tendency to hæmorrhage dependent upon an inherited taint of phthisis.

In one of the most remarkable cases of pulmonary hæmorrhage that has come under my notice, remarkable because of the trivial causes sufficient to induce profuse blood-spitting, I failed to establish any ancestral taint from direct history, the connection only being proved by the condition of two sisters of the patient, who suffered from scrofulous glands, and of one cousin who suffered from hæmorrhage from the bowels and epistaxis.

In another case of initial hæmorrhage the history of the parent and grandparents at first excluded the idea of inheritance, a fact subsequently established by a closer investigation of the ancestral history, the great-grandfather having died of phthisis.

The direct evidence of phthisical taint was not obtainable in the two following cases, but the inherited predisposition to hæmorrhage appears as a family condition:

Case XLI.—Thomas M——, age 26, carpenter.

No history of phthisis obtained.

Father now 56 years of age. He has no symptoms of phthisis, and is in good health, but he has been accustomed to spit blood, half a pint at a time, as long as the son can remember, recurring at intervals.

Patient was working at his trade in 1874, at the age of 22, and spat blood to the amount of a pint and a half. This recurred in May 1875, and again in June 1878. He has been under my care four years, and has not yet developed signs of phthisis.

Case XLII.—Robert W——, age 24.

Father and mother alive—no history of phthisis.

The patient, while coughing, spat up half a pint of blood twice. Gums much inclined to bleed when brushed. No symptoms of phthisis.

The rest of the family (three in number) suffer from profuse epistaxis. No evidence of phthisis.

In this category of cases I had originally included one which I was fully persuaded belonged to the same class, although I at first failed to elicit from the patient any account of inherited disease.

Case XLIII.—Martha B——, age 30. Married; no children.

No history of cough, nor of any tendency to bleeding, was obtained as regards the parents.

She applied to me for advice regarding copious attacks of bleeding from the lungs. The catamenia were perfectly regular, but rather scanty.

The first attack of bleeding occurred in consequence of a trivial exertion. She had been playing with some children and pulling down the branch of a mulberry-bush for them. During the subsequent night she coughed up a large quantity of blood; and since that time she had frequent recurrence of the bleeding, sometimes raising as much as two pints. These attacks generally came on just before or just after the catamenial periods. There were no signs of phthisis.

After some investigation, she at last brought me a letter from her mother, in which it was stated that the mother's father, the patient's grandfather, suffered very much from profuse epistaxis, while his brother suffered also from similar attacks, and actually died from epistaxis at an advanced age.

Since my attention has been directed to the necessity of investigating the inherited tendency, in all cases of bleeding I have rarely failed to establish the connection in cases where the bleeding has been copious, has occurred during the early stages of pulmonary disease, or more especially before any pulmonary disease could be detected, and where it has been due to some very trivial exertion.

The same conditions are observed in the transmission of this special form of bleeding as in the more intense form of hæmophilia, and the two conditions appear to be closely linked together, the one being a modified form of the other; whether they are identical conditions differing only in degree is a point which is open to doubt.

Profuse hæmoptysis affects men more frequently than women, and in a greater degree.

In Pollock's analysis of 351 cases of profuse hæmoptysis, 267 occurred in males, 84 only in females—a proportion of three to one.

In hæmophilia the difference is greater, and is put down by one authority at seven to one.

Both diseases affect women with a lesser degree of intensity

than men, and the danger to life from either disease is infinitely less among women. Out of fourteen cases of fatal hæmoptysis which I have lately examined, two only were women.

In many cases of blood-spitting, sanguineous fluxes occur from other parts of the body; in some instances this is no evidence of the tendency to bleeding during the early period of life. The subsequent development of this tendency during the period of growth may with some plausibility be ascribed to imperfect structural growth at the time; the greatest strain being thrown at these periods on the thoracic organs, and particularly on the lungs.

In putting forward this theory regarding the inherited tendency to hæmoptysis, I reserve my opinion as to how far this tendency is identical with that established in hæmophilia—what I have endeavoured to show is that the same laws of transmission are observed in both cases; future observations are required to establish the identity of the two diseases from a pathological aspect.

The statistics indeed of hæmophilia go to show that this form of bleeding is not connected with, and does not terminate in, phthisis; but its relation to scrofula is insisted upon by more than one observer, so that a conjecture might be raised to this effect, that the tendency to hæmorrhage which in small degree results in bleeding from the lungs, may by continued inheritance be ultimately developed in an intense form more generally, while the tendency to phthisis is gradually lost.

So many cases of this class of hereditary bleeding have come under my observation as to afford convincing evidence of the truth which I have endeavoured to establish, hereby removing this class of cases from an initial connection with tubercle, a point of the greatest importance, inasmuch as much consolation may be procured for the patient by showing the evident connection between hæmorrhage from the lungs and other bleedings to which much less importance has always been attached.

I must remark that negative histories should be carefully sifted, and the question carefully threshed out, as I have often been met by a distinct denial from the patient of any heredity which has subsequently been established by careful investigation of the family history.

CHAPTER VII.

PHYSICAL CAUSES OF HÆMORRHAGE.

Physical causes which are mechanical in their action are often ultimate agents of hæmorrhage in many cases, the chief difficulty with regard to each case being the exact diagnosis of the proximate pathological condition present at the time.

The influence of exertion in the ætiology of blood-spitting has not received so much attention as it deserves from the importance and frequency of the event, for cases occur in which hæmoptysis has followed some unusual exertion in which no positive proof of pulmonary disease can be shown to exist; but in no way daunted by this difficulty, authorities have recurred again and again to a theory of latent tubercle, which to them accounts for everything. Probably every medical man has observed such cases, without history of pulmonary disease, without present symptoms, and without any subsequent untoward result, although under observation for a long period. Such cases have come under my own notice, and the physician's reputation is not likely to be increased by a diagnosis of tubercle, inasmuch as no tubercular condition is proved to be present, nor becomes subsequently developed. The true interpretation of these cases appears to depend simply upon the relative strength of the pulmonary structure, and the force which is brought to bear upon it, and the more delicate the organ the more trivial the exertion required to induce the untoward result.

Muscular exertion, as regards its effect upon the thoracic organs, may be considered from three points of view:

1. Those forms of exertion, which call into play chiefly the muscles of the arms and thorax—the lifting of weights, striking heavy blows, pushing, &c.—require often the utmost strain on

the part of the individual, generally of short duration but excessive.

2. Those actions in which the muscles of the lower part of the trunk are used, as in walking and running, exercise which requires generally prolonged exertion.

3. The combined action of the muscles of the body, as in the carrying of weights, &c.

Momentary exertion of the first class, if in excess of the individual's powers of resistance, results in overstrain of some weak part of the body, and may cause rupture of the cardiac valves, or of some pulmonary vessel.

Prolonged exertion in walking and running leads to excessive hyperæmia of the lungs, consequent upon the rapid emptying of the veins into the right side of the heart, and either the lungs give way under a condition of general congestion with extravasation of blood, or local apoplexies of a similar kind occur; or, on the other hand, the heart may become overstrained, and give way in a condition of general dilatation.

The combined exertion of walking and of lifting or carrying weights gives rise to similar conditions and to aortic aneurysms.

In cases of the first kind immediate rupture takes place from an inability to support the strain; in the second class of cases diapedesis may be the source of the bleeding, or rupture may take place secondary to a congestion. Of this latter condition three instances are given in a previous chapter showing that congestion does occur from exertion.

I have never seen nor read of a fatal case occurring directly after exertion in a previously healthy individual.

When bleeding occurs as a result secondary to pulmonary disease, some proximate cause, from exertion, excessive heat, or shock, may often be established, a delicate structural condition being induced by the pulmonary disease; and so many cases of phthisis occur even among males without any indications of bleeding throughout the whole course of the disease that in all cases in which blood is expectorated the closest inquiry should be made as to the proximate cause.

Case XLIV.—A man who had been under my care for many months with old bronchial and pulmonary disease without the

slightest indication of bleeding, astonished me one day with the information that he had a few days before expectorated a large quantity of blood. On inquiry I found that he had, after a very cold drive, helped to lift a heavy box to the top of his cab. He has since had no recurrence of the attack.

The following cases may be quoted as examples of hæmoptysis immediately following upon a strain:

Case XLV.—Alfred H——, age 30.

No hereditary predisposition. No previous illness.

Three months ago was walking fast, and found his mouth full of blood—lost more than a pint. Felt faint.

Hæmoptysis returned with the exertion of getting into bed.

Physical signs.—Deficient respiratory murmur.

Crepitation of blood in right infra-spinous fossa.

Case XLVI.—William V—— age 31.

Father died from hæmorrhage due to strain. No history of cough.

Six weeks ago hæmoptysis to the extent of a quart while loading a cart with gravel. A broad-chested man. Had suffered from cough two weeks previously.

No physical signs.

Case XLVII.—Henry E——, age 42.

Father asthmatic.

No cough previous to an attack of hæmoptysis, which occurred thirty-three months ago, while running up a flight of stairs. Hæmoptysis profuse.

Peculiar chest, narrow and long.

Case XLVIII.—Hilton W——, age 18.

Father died of dropsy.

Four months ago was walking home from a theatre when profuse hæmoptysis came on. Slight previous cough.

Chest long and narrow.

Such cases as these are not so frequent as examples of hæmoptysis occurring some hours after the strain. In the first case no evidence of cause except the strain could be obtained.

In the second case there was some slight cough before. In the other two cases the peculiar conformation of the chest seems to point to the constitutional imperfection which may

have encouraged hæmoptysis: possibly the length of chest tended to prevent free expansion.

The following examples may be given of hæmoptysis occurring some hours after strain:

Case XLIX.—John B——, 31. Baker.
Mother died of rheumatic fever.
No predisposition to phthisis.
Was carrying a sack of flour, and in one hour after spat up two quarts of blood.
Broad-chested and very muscular. Had some time ago a cough.
Physical signs.—Crepitant sounds of blood at left apex.

Case L.—James P——, 38.
No hereditary predisposition. No previous cough.
Four years ago was lifting a puncheon of rum (2 cwt.), on the following day hæmoptysis to the extent of one pint.
Had been accustomed to drink much.

Case LI.—Edward M——, age 23.
No hereditary predisposition. No previous illness.
Had been running in a foot-race, and some hours afterwards was attacked with copious hæmoptysis.
The same thing occurred two years after on lifting a heavy weight.

This case I have recorded more fully on another page. In individuals who inherit a tendency to bleeding the exertion necessary to produce such a result is sometimes very trivial. I have known bleeding to occur profusely from jolting in a railway train, from reading aloud, from a fit of passion, and from reaching over a table for a plate. Slight causes should at once raise suspicion of inherited weakness, and the family history should be carefully investigated.

Opinions are much divided among those who have written on the subject of pulmonary disease regarding the effects of the use of wind instruments. Pollock says on this point: 'We have had some cases at the hospital which seemed to favour their use, but the impression left was that the practice was often productive of hæmoptysis. We should hesitate to recommend it

believing that the same amount of benefit may be obtained by some other modes' (p. 389, 'Elements of Prognosis in Consumption').

This last sentence evidently refers to the advantages to be derived from free exercise of the lung. On looking through my case-books I find comparatively few players of wind instruments, and I conclude that pulmonary disease must be rare as a consequence of the practice. Many of the patients whom I have examined (and recorded in the Appendix) had been playing for some years before the pulmonary disease was developed, and in some it appears to have followed the disuse of the instrument.

As regards the bearing of this practice on the causation of hæmoptysis, it is not very evident how it can cause an increase of intravascular pressure: it may by exhausting the powers of the individual lead to impairment of the circulation and consequent hæmoptysis, as Pollock suggests; but this does not appear to have been a frequent result in those cases of which I have given the details.

The following case might be taken to be evidence in opposition to the opinion just given, but I cannot accept a single case of the kind as decisive:

Case LII.—Harry J——, 32, was admitted as an out-patient under my colleague, Dr. T. H. Green, in 1877.

He was a trombone player, and had raised a pint of blood three years before attending the hospital.

The note of the physical signs in February of that year was that obscure crepitations were to be heard at the left lung. He remained under Dr. Green's care until August 1878, up to which time there was no recurrence of bleeding. The man continued playing the trombone in the orchestra of a large London theatre until August, when he was seized at the end of the performance with copious bleeding, which terminated fatally, the man dying in the orchestra.

If this result was caused solely by the exertion of playing, why was bleeding absent for four years, during which time he followed his usual vocation? It seems fair to suggest that the heat of the theatre may have had also something to do with the fatal bleeding.

In contrast to this case, let me give the details of another

patient, who is now under my care, and who was the fellow trombone player of the above patient in the same theatre.

Case LIII.—James R——, 41. Trombone player for twenty years. Was in the Coldstream band, and invalided eight years ago for phthisis. For the last five years he had spit blood occasionally in small quantities—only a teaspoonful at a time.

On examination I found that he had a hard dry cavity of old standing in the upper lobe of the left lung.

He stated that the use of the instrument caused him little exertion, and no distress, and he did not attribute the bleeding to it. He had been playing every night in the theatre for many years, and depended upon his instrument for his livelihood.

In considering the possible effect of such exertion on the patient, previous training must be taken into account, as long practice enables a skilled performer to do his work with the least possible strain. I have one or two instances (in my notes) of bleeding following the exertion of singing or recitation, but these causes are not effective unless the tendency to hæmorrhage is very great.

In some cases the exercise of the lungs consequent upon their forced inflation appears to have really postponed the development of pulmonary disease, which has been established when the instrument has been laid aside. Those that are curious on this point may refer to the Appendix, where they will find details of the cases which have come under my notice.

Intravascular pressure may be increased to a great extent by beer drinking, and I have described the conditions which occur from this cause. The external appearance of the beer drinker or toper cannot be mistaken: the individual (of either sex) is corpulent, bloated, has a red congested face, the veins full, especially those of the extremities and the hands, the breath tainted with the peculiar odour of beer, the heart rapid and feeble in action, often dilated, digestion impaired, the bowels frequently relaxed, the urine copious and lithatic, with a general tendency to capillary stasis and purpura. In marked cases degeneration of the vessels is induced, which results in a very facile condition of bleeding, and is correspondingly difficult to arrest. When once pulmonary disease is established in these cases, if the bleeding

recurs, as it is very apt to do, there is very little prospect of recovery, and the patient rapidly goes from bad to worse.

I have already given at p. 39 the details of a remarkable case in which symptoms closely simulating those of hæmophilia were engendered by copious beer drinking in the case of a cellarman; here treatment was successful in restoring the patient to a condition of health. The following is another similar case, in which phthisis had occurred before the man was seen, and it was impossible to stay the downward progress of the disease:

Case LIV.—James C——, age 33. Tailor.

Father and mother without cough; mother died of ulceration of bowels. No hæmoptysis.

One brother and two sisters died of consumption and hæmoptysis.

This man had been an extensive beer-drinker, and had all the appearance of alcoholism. He had suffered hæmoptysis, copious, three times since Christmas, nine months before he applied. He was then emaciated, and had cough.

The hæmoptysis was very obstinate in spite of remedies, and it continued, though not very profuse, for one month. He also suffered from dyspepsia and diarrhœa, which very much aggravated his symptoms, and prevented recovery. Also he had very sleepless nights. He lost flesh very fast. When he first applied, I found only symptoms of destruction commencing at right apex, but these gradually increased, and in two months cavities were found in both lungs, with corresponding emaciation.

At the end of November he again had hæmoptysis, and from this time he became rapidly worse, and too ill to continue his attendance at the hospital.

The two agents in the causation of blood-spitting just described are sufficiently powerful of themselves, without the presence of pulmonary disease, to produce bleeding; but the third agency, to which I shall direct attention, does not, so far as I am aware, cause pulmonary hæmorrhage under the ordinary conditions of life, unless there is a special tendency to vascular rupture from the presence of phthisis or other pulmonary disease.

It is impossible for me, therefore, to appeal to the evidence

of isolated cases to prove its effect, and it is only by the tabulation of a number of cases that cumulative evidence of its great influence is shown.

The main effect of heat on the body is the acceleration of the pulse, which, under some conditions of great heat, has been doubled. This results from the expansion of the circulating fluids, and the consequent increase of intravascular pressure. The increased size of the vessels is shown in ordinary life, under the effect of hot weather, by the swelling of the hands or feet, which is recognised by the difficulty of putting on or taking off rings, gloves, or shoes. It would appear also, from experiments that have been made by C. B. Williams, that the tonicity of the arteries is effected by heat, and the diminution of this quickly leads to an enlargement of the calibre of the vessel, and a relaxation of the blood stream; in the same way the tonicity of the veins is affected.

Added to this, it appears probable that the extravascular pressure is diminished by the admission of heated air, in consequence of the less expansive power which results from the subsequent heating in the lungs. Cold has exactly the contrary effect.

The season at which hæmoptysis is most rife is during the summer heat; and I have drawn up a table of 1,000 cases in which the date of the blood-spitting was accurately known. This table will be found in the chapter on Statistics, and it shows the very large preponderance of cases occurring during the summer months; *per contra*, there is considerable diminution during the winter months.

Alternations of weather are shown also as effective, especially during the spring winds and the foggy damps of November. The results of this table are extremely suggestive as regards the climatic treatment of hæmoptysical patients.

In order to show the auxiliary effects of the hot weather of the summer months, I will quote a rather remarkable case, in which blood-spitting occurred at particular times:

Case LV.—Eliza W——, age 36.

Had suffered from cough altogether eight years before she came under my care in 1873. I then detected a cavity in the right lung.

She first suffered from an attack of blood-spitting in November 1872.

She remained under my observation, except during the year 1876, until her death in November 1878, which took place from a fatal attack.

Blood-spitting occurred in May 1874, 1875, 1877, 1878.
June 1875,
July 1873, 1874, 1878.
Aug. 1878.
Nov. 1878. Fatal.

A sudden increase of temperature has been observed to be followed by frequent attacks of hæmoptysis affecting several patients at the same time in the hospital, and a remark to this effect is made in the Second Medical Report of the Hospital.

Recently such an increase of temperature took place, severe frost yielding rapidly to a thaw and much warmth. It was observed that several patients were attacked with hæmoptysis, and one unexpected attack ended fatally at the same time.

Attention deserves to be especially directed to this condition in the ætiology of bleeding, inasmuch as it shows the necessity of careful attention, not only to the kind of climate most suitable for these cases, but to the temperature which should be maintained in the dwelling-rooms.

Of the effects of severe cold in producing blood-spitting, I have no examples to record, nor has any marked instance of shock come under my notice; such occurrences are rare, but the three agents of which instances have been given are frequently exerting their influence, and must be looked upon as potent in action.

Some practical deductions may be made from these considerations as to the treatment of blood-spitting: the absence of exertion, the diminution of ingesta, and the prevention of bleeding by a cold dry temperate climate, are important points in the treatment of these cases; and the remedial effects of styptics are materially increased by strict attention in the warding off from the patient any of those forces which might tend to aggravate the bleeding.

CHAPTER VIII.

THE CLINICAL ASPECTS AND SYMPTOMS OF BLOOD-SPITTING.

When blood is ejected from the mouth, the first anxiety of the patient is to know the source from which it has proceeded, inasmuch as, following the vulgar notion with regard to blood-spitting, the patient is very apt to consider himself doomed to an early grave if the physician finds that the blood proceeded from the lungs. It is, therefore, to this point that the attention of the physician is first directed, and a diagnosis is made from the appearance of the blood, macroscopically and microscopically, and from an investigation of the actual condition of the air-passages and thoracic organs.

There are certain well-known indications which in some cases of scanty bleeding will sufficiently indicate the source of the blood: as, for example, in cases of oral bleeding, occurring in hysterical patients—the blood is mixed with much saliva, and has a peculiar viscid streaky appearance, which is well known to every practical physician; in like manner the blood from the nares can be detected in many cases by the peculiar admixture with the nasal mucus; but in cases of copious bleeding in which the blood is unmixed, very great experience and care is required for a true diagnosis.

In the lecture which is devoted by Graves to the consideration of hæmoptysis, this passage occurs, remarkable for its boldness and truth:

'You are told gravely, that you can distinguish blood discharged from the stomach from that which is discharged by the lungs by the differences of its colour and consistence, and the presence or absence of air bubbles. No, gentlemen, you cannot.' ('Clinical Lectures,' vol. ii. p. 130.)

This statement may be illustrated by an appeal to such

clinical experience as the following:—Two patients died of a sudden attack of fatal bleeding from the mouth and nose; in both cases large quantities of light red blood poured out of the mouth. At the post-mortem examination it was found that death in one case proceeded from ulceration of the lesser curvature of the stomach, in which was situated a small aneurysm of the coronary artery of the stomach: the vessel had given way and caused death. In the other a small aneurysm of the pulmonary artery was found in the lung, the rupture of which had proved fatal. There are broad distinctions which in many cases may help to form a diagnosis, as, for example, the presence of blood in the evacuations; but even this cannot be considered as an unfailing proof of blood having proceeded from the digestive tract, inasmuch as blood is often swallowed, and is found in the stomach in cases of fatal blood-spitting. Many authors have decided, quite erroneously, that blood from the lungs is always of a dark colour, forgetting entirely that the blood of the pulmonary artery is changed from dark red to arterial red on exposure to air in the lungs, and those that have any experience of bleeding from the lungs know full well that the blood raised in large quantities is often of a bright arterial red. If blood be retained for any period in the lungs, it loses its arterial colour and becomes dark; and I have seen blood from an aortic aneurysm which could not be distinguished during the life of the patient from that arising from a condition of congestion. Dark blood is sometimes raised unaltered in colour when a large branch of the pulmonary artery is ruptured.

If the quantity of blood expectorated be slight, diagnosis of the source from which it has proceeded can only be obtained by the indications which may be afforded by the microscope, the most important of which is the presence of elastic lung tissue in the sputa.

The first object of the physician is to allay the patient's fears, and although this may be done by a statement that the blood comes from the throat, and not from the lungs, the most truthful consolation that can be given is this: that bleeding occurs frequently without the presence of pulmonary disease; that its exact source may not be determined in every case, but that evidence of consumption is not established by its appearance only.

For women, the most anxious of patients, the prognosis is more favourable than for men, inasmuch as menstrual irregularities are a very fertile source of pulmonary bleeding, from which no serious results need be anticipated. This is the general opinion of writers on this subject, and many cases have been from time to time recorded in which vicarious menstruation has occurred regularly for years without impairment of health. A remarkable case from Pinel is quoted by Sir T. Watson, in his Lectures, of a woman who menstruated through her lungs at each monthly period from her 16th to her 58th year, that is, during 42 years of her life. As Watson remarks, 'cases of this kind are not all uncommon, although the vicarious hæmorrhage seldom persists so long and so steadily. They are not usually attended with any peril to life.' West, however, records that he has had experience of such a fatal case, presumably from the amount of blood lost.[1]

The immunity from secondary results enjoyed in these cases may be explained partly from the fact of the blood proceeding from lungs which are not occupied by disease, partly from the gradual flowing of the blood which generally occurs in these cases, and it may be also from the disinclination to clot, which is a peculiarity of the menstrual blood, and may be shared by the blood generally at this period.

That menstrual blood contains no fibrin has been shown by Vogel, and confirmed by Schmidt, as quoted by Lehmann in his 'Physiological Chemistry'; but I am not aware of any analysis showing the condition of the blood generally at this period.

Vicarious menstruation, like menorrhagia, occurs in women under two opposite conditions as a consequence of general debility interfering with the regular discharge of a natural function, and as a result of plethora in which there is increased intravascular pressure from constitutional conditions increasing the quantity of the circulating blood.

In the former class of cases the amount of blood raised is generally small, while in the latter class the amount is often very profuse. The treatment will of course depend upon the genera constitutional indications which accompany the bleeding.

[1] 'Diseases of Women,' p. 44.

In some instances of hæmoptysis casts of the bronchial tubes have been ejected formed of the fibrin of blood.

An interesting case is given by Flint in his work on Phthisis (p. 105). The casts in this case represented tubes of the size of the second or third subdivisions, and were formed of fibrin, and red and white blood globules.

Another case of a similar kind is recorded by Graves in his 'Clinical Lectures' (pp. 146), which is so interesting that it may be given in his own words:

Case LVI.—A gentleman who had been ill for many days with blood-spitting, and had been often bled, was much exhausted. I had visited him in the morning, and had just left him when a fresh burst of blood took place. Contrary to my orders, he was again bled, and when Dr. Stokes arrived, in about three quarters of an hour afterwards, he found him collapsed, almost asphyxiated, and struggling for life.

The right side of the chest expanding and contracting energetically, the left almost fixed and motionless.

Dr. Stokes immediately changed his position and gave him a glass of wine, when he made one more effort, and violently expectorated a coagulum, consisting of fibrin, in some parts nearly colourless, forming a complete solid mould answering to the left bronchus and its ramifications down even to some of the minuter tubes.

The mode of death varies in different individuals, as indicated by the condition of the heart. It may occur from fainting, dependent upon the quantity of blood lost, or upon weakness of the heart, the result of feeble muscular power, or of actual disease, fatty degeneration of the walls, valvular disease, or atheroma of the vessels. The heart under these conditions will be found uncontracted and full of blood, the right side especially being crammed with dark blood.

The disposition to faint is not always proportionate to the amount of blood lost.

Death may occur from suffocation, from the regurgitation of blood into the tubes, which the failing or feeble powers of the patient are not sufficient to remove. In such an event the heart continues to beat for some time after breathing

has ceased, and is generally found semi-contracted and empty.

But there is another condition under which death may occur. Comparatively little blood may be raised, and the lungs may be in a fairly healthy condition, but the balance of life may be upset by the rupture of a small vessel; in these cases it seems probable that death is due to the sudden cessation of respiration, not as the result of suffocation, but possibly of suddenly altered tension in the pulmonary circulation. In these cases the lungs fail to deliver blood to the left side of the heart, which is found closely contracted and quite empty.

In brief the occurrence of blood-spitting may be due to the following conditions:

1. A tendency to hæmorrhage, the result of some special constitutional delicacy hereditary from a parent or progenitor affected with a like tendency.
2. Over-exertion, which may result in immediate or deferred rupture; the amount of exertion necessary for the result is dependent upon individual peculiarities.
3. Congestion arising from exposure to cold, feebleness of circulation, or copious potations.
4. Alterations of the blood, as in scurvy, purpura, and the eruptive fevers.
5. Ulceration and aneurysm of the pulmonary vessels.
6. Thrombosis and embolism of the pulmonary vessels.
7. Ulcerative destruction of excavation involving new formed vessels.
8. Bronchitis arising from inflammation and the passage of irritating fluids over the tubes.
9. Pneumonia.
10. Menstrual irregularities.
11. Cardiac disease.
12. Diseases of the liver.
13. High temperatures.
14. Presence of pulmonary tumours and carcinoma.
15. Syphilitic visceral disease.
16. Aneurysms of the systemic circulation.

To this list may be added a supplementary one of those

diseases in which blood-spitting rarely occurs, or arises as an independent occurrence:

Pleurisy.	Acute phthisis.
Emphysema.	General pulmonary tuberculosis.
Pneumothorax.	Scrofulous phthisis.
Œdema of the lungs.	Fibroid disease.

The symptoms preceding an attack of blood-spitting are generally of a uniform character. Patients that have had one or two attacks are generally forewarned, and anticipate the event with some certainty. The symptoms of which complaint is usually made are a feeling of tightness across the upper part of the chest, or a feeling of weight accompanied with some dyspnœa and incapability of exertion. This is followed by a sensation of irritation of tickling about the fauces, and by a salt taste in the mouth, which generally heralds the actual attack.

During the attack considerable anxiety is frequently evinced, but this is not always the case. The pulse is excited and bounding, partly from the excitement of the moment, partly from the relief of intravascular pressure, and the respiration is hurried and rapid. If the attack is profuse, the patient becomes blanched and faint, and the pulse becomes weaker, but a very large quantity of blood (several quarts) may be sometimes lost without any permanent result to the patient.

At other times a very small quantity may suffice to cause death, and death may even occur from fatal bleeding into the lungs without any outward and visible sign of the event. An instance of this kind is recorded by Rasmüssen in his valuable contribution to the literature of Pulmonary Aneurysm. A patient suddenly fainted and died without indications of blood-spitting, in whom after death the presence of a ruptured pulmonary aneurysm was established.

As an instance of profuse pulmonary bleeding, the case given by Pollock may here be cited:

> The largest quantity recorded was eight quarts, by a man, aged 20, in the second stage of phthisis, occurring within the first three months of illness, and whose case was under observation for three years, during which time the hæmorrhage was frequently renewed.

Thermometrical observations regarding pulmonary hæmorrhage.—The evidence of pyrexia following attacks of blood-spitting is to be obtained by the use of the thermometer, and there is a good deal of difference observed in different cases. In a number of cases hæmoptysis copious in amount may occur without any very decided alteration in the body temperature; occasionally it is followed by slight reduction, and again, in other cases, pyrexia may set in and daily exacerbations may be observed for some days after.

Dr. Wilson Fox, in his paper on the 'Temperature of Phthisis,'[1] has the following remarks on the subject:

'Hæmoptysis, as I have more than once observed, even when the amount of this has been considerable, does not appear to exert any influence on reduction of temperature. I have known the temperature rise on its occurrence, and I have also known the temperature fall before the hæmoptysis has completely ceased. In some cases no fever follows a first attack of hæmoptysis, and very high pyrexia may be observed after a second attack.'

The subject appears to me a very complex one, and alterations of temperature arising from blood-spitting must, in some measure at least, depend upon the following points:

The presence or absence of pulmonary disease at the time.

The purity or admixture of the blood (as regards septic matter) which regurgitates into the lungs.

In a case for the observations of which I am indebted to Mr. Evelyn Rich, taken five times a day for nine days, during several attacks of profuse bleeding, the following alterations were noted:

Case LVII.—At 6 p.m. the temperature was noted at 100° F.; a profuse attack of bleeding occurred at 7 p.m. to the extent of a pint; at 9 p.m. the temperature was found lowered by $\frac{1}{5}$ of a degree.

The same depression occurred again, but to a greater extent.

The temperature at 6 p.m. was 98·8° F.; at 7 p.m. bleeding recurred to the extent of half a pint, and at 9 p.m. the temperature was lowered $\frac{3}{5}$. The temperature was depressed for some days, and did not regain the previous standard for five days. These obser-

[1] Med.-Chir. Trans. lvi. 399.

vations were made in a case of very advanced phthisis, and the depression in temperature is a simple and intelligible result of the loss of blood.

In the following case, however, for which I am indebted to the kindness of Dr. Powell, the marked pyrexia following two attacks of bleeding is capable of more than one explanation: it may be due to exacerbations of irritation in an already diseased lung; it may be due to local infection from the admixture of septic matter from the source of hæmorrhage with the blood landed in the lungs by insufflation.

Case LVIII.—William P——, age 23. Suffered from cough for three years. Hæmoptysis twelve months ago first; quantity of blood two to four ounces.

Physical symptoms on admission in May 1878:

Cavity at left apex.

During the months of May and June the temperature of this patient was taken regularly twice a day, and it varied between 97·8° F. and 99·6° F. The general temperature observed for several days being between 98° F. and 99° F.

On August 15 he began to spit blood, and continued to spit blood, in quantities varying from two to ten ounces, until September 16, the blood being raised for twenty-two days.

At first a depression of temperature was observed up to the morning of August 25, at which time it was noted at 97° F. From that time very marked exacerbation took place, with nocturnal high temperatures, gradually increasing, and ranging on August 31 to between 96° F. in the morning and 104·2° F. at night—the greatest height being reached on the night of September 7, when it nearly reached 105° F. The temperature then, after the lapse of some days, returned to its normal condition, and during October remained fairly steady.

At the beginning of November the patient was again attacked with blood-spitting for four days, the greatest amount being 10 oz. During this time the temperature remained quiet; but on November 5 exacerbations were again noted, with temperatures ranging between 98° and 105°, returning again in the middle of December to the normal range, at which it remained during January.

The explanation of this interesting case must be hypothetical, but the interpretation that seems at least probable is that

the blood was mixed with some of the secretion from the cavity, thus creating irritation in the tissue, which was probably at the time in a hypersensitive condition.

These two cases indicate the variations which may result from bleeding, but the latter instance is rare. Many cases occur in which no variations of temperature take place, and in them it is fair to conclude that no great harm has resulted from the loss of blood, nor any irritation been set up from its presence in the lungs.

Sometimes an elevation of temperature for a short period is observed immediately after the bleeding, which may be due to the alarm caused by the sight of blood; in such cases the temperature soon sinks to its usual standard.

CHAPTER IX.

PHYSICAL SIGNS.

The pathological conditions which follow the presence of blood in the lungs having been thus set forth, the ground is cleared for the better appreciation of the sounds which are heard in cases of hæmoptysis.

Those who are still sceptical will not be convinced by any description of sounds which cannot be accepted as evidence of so decisive a nature as that already adduced. The careful auscultation of a few clinical cases would be more likely to lead to conviction than any number of cases that can be given on paper, and any description of sounds, however elaborate, may be passed by, while each critic may assign his own interpretation to the description. The experience of a large number of cases convinces me that the physical signs are peculiar in kind in successive stages of hæmorrhage, and they support the theory already advanced on pathological grounds.

After an initial hæmoptysis of any extent occurring without a history or symptoms of previous pulmonary disease, the presence of blood in the lung is indicated by a fine moist crepitation of a peculiar kind, coarser than the crepitation of pneumonia (which is a standard sound), and more viscid than that of œdema.

The authority of Laennec may be quoted to show how fully aware he was of the peculiar characteristic sound of blood in the lungs. In discussing the signs of what he terms hæmoptoic engorgement, he says that one of them is 'un râle crépitant qui, aux environs du point où la respiration ne s'entend pas, indique la légère infiltration sanguine.

'Ce râle crépitant a toujours lieu au début de la maladie: plus tard il cesse souvent de se faire entendre. Quand une hémoptysie présente ces signes, on peut affirmer que le siége de l'hémorrhagie est dans le tissu pulmonaire et non pas simplement dans les bronches. Cependant, de même que dans l'hémorrhagie bronchique, on entend vers la racine des poumons surtout un râle muqueux à grosses bulles, dont la matière paraît plus liquide et dont les bulles semblent plus grosses que celles qui sont formées par de la mucosité: leurs parois semblent plus minces et elles crèvent plus souvent par excès de distension. Le bruit de cette rupture se fait entendre d'une manière non équivoque.' [1]

I have given this quotation at length because it is an admirable description of the sounds in question, and one which could not be surpassed.

The only point in which I venture to differ is this, that the peculiar sound is heard over the parts of lung into which blood has been inhaled, and the sounds do not localise necessarily the source of the hæmorrhage.

Laennec points out that it is heard often near the root of the lungs, and the most usual place, according to my experience, is just above the root of the lungs; this is not the usual locality for congestion as we know it in the post-mortem room, nor can the signs be mistaken for those of congestion, inasmuch as they differ in kind and in subsequent alterations.

Two or three cases may be given from my note-book as examples, and many more could be added to them. In a number of patients applying for advice after a recent attack of copious hæmoptysis I have found these signs in some part of the lungs, usually the upper lobe, unless pulmonary disease has been previously present.

Case LIX.—Sarah C——, age 40.

History of hereditary predisposition to hæmorrhage.

Used to suffer epistaxis when young profusely.

Nine months before visit spat up a pint and a half of blood, and when examined was found to be suffering from emphysema and bronchitis. Hæmorrhage again recurred to the extent of a quarter of a pint a month after the first examination, and signs were found

[1] 'Traité de l'Auscultation Médiate,' p. 372.

immediately after the blood-spitting, showing the presence of blood under the left clavicle, viz.: peculiar crepitation heard at the end of inspiration. This was not present at the first examination.

Case LX.—Isaac M——, age 18. A clerk. July 23, 1873.

Father had a cough.

A sister } died of phthisis with hæmoptysis.
A brother }

A sister has phthisis without hæmoptysis.

A brother used to profuse epistaxis; no phthisis, no hæmoptysis.

Patient has not suffered from epistaxis.

Hæmoptysis twelve months before, in 1872—quantity, a handkerchief full.

Was spitting blood at time of visit.

Physical signs.—Fine moist crepitations at left apex.

He again applied for advice in December 1878; he had spit blood, a teacupful, in July 1878, and again seven days before visit. I found the same kind of crepitation a little lower down, over the left upper lobe; but he was not worse, had not lost flesh, and there was no reason to believe that he was suffering at the time from phthisis.

This is six and a half years after first date.

Case LXI.—William V——, age 31. Carman. May 10, 1878.

Father died through breaking a blood-vessel from lifting.

No history of cough.

Six weeks before visit was loading a cart with gravel, and he was seized with hæmoptysis to the extent of a quart. He had a slight cough for two weeks before. No other illness. Broad-chested.

Physical signs.—Right lung, dull percussion from clavicle to third rib, with spongy alveolar crepitation.

Left lung.—Less crepitation under clavicle, and in region of nipple.

Case LXII.—Alfred H——, age 30. May 1878.

No hereditary predisposition.

Three months before visit was walking fast, and found his mouth full of blood: lost over one pint, and felt faint from loss.

It recurred with exertion of getting into bed.

Never had any cough.

Physical signs.—Slight dull percussion, with deficient alveolar respiratory murmur; inspiratory high-pitched, expiratory short.

Occasional peculiar viscid crepitation at end of expiration over the supra-spinous fossa.

Case LXIII.—Albert White, age 17. Joiner. August 6.

No hereditary history obtained.

Hæmoptysis to the extent of a pint and a half three weeks before visit.

Physical signs.—Peculiar alveolar crepitation in right supra-spinous fossa.

September 17.—These sounds were replaced by imperfect expansion and dull percussion, with high-pitched respiratory murmur.

Case LXIV.—Emily B——, age 24; single.

Has never suffered from cough.

Examined first in February 1876. No evidence of pulmonary disease.

Suffering from dyspepsia. Catamenia scanty.

No subsequent cough. In September 1877 had hæmoptysis, and spat up three pints of blood. No cough at the time. She applied for advice at the end of January 1878.

Physical signs.—I then found a depressed condition of first space on the right side, with increased voice resonance and high-pitched expiratory murmur. No evidence of phthisis.

Case LXV.—Henry E——, age 42. Smith. Applied as out-patient in January 1878.

Father's family subject to cough.

Father lived to 63. Died of asthma.

One sister with bad cough.

No epistaxis. Suffers much from piles.

Thirty-three months before was running up stairs and was then attacked with hæmoptysis (several mouthfuls).

Never had any cough.

Five months before visit was hurrying to Victoria Station.

Physical signs.—High-pitched percussion with general scattered patches of crepitation, with an emphysematous condition between and in neighbourhood. The same condition at both apices, and also in the anterior portion of left base.

Case LXVI.—A young man, age 20. Carpenter by trade. Consulted me first in March 1877, and the following history was obtained.

He had never had any illness, never suffered from cough, and

no history of inherited predisposition to cough or hæmorrhage could be elicited.

He had always been a fast runner, and first ran for money in January 1875. For three months he ran races. In April 1875, just before the day of a race, after a trial race he was seized with hæmoptysis and spat a pint of blood.

He was admitted under Dr. Powell December 28, 1875.

The following physical signs were then noted:

Right apex slightly less expansive. Percussion generally resonant in front. Respiration harsh and wavy, with some dry crackle after cough to nipple.

Heart's action excited, impulse exaggerated, situation normal. Systolic bruit heard over the pulmonary artery.

Before the man left the hospital in March the following notes were made:

Right apex then found dull on percussion; creaking at the end of inspiration; and at the second space a peculiar metallic clicking râle was heard at the end of deep inspiration.

Soon after this he applied to me, and I found on examination the same signs. He remained under my care only a short time, as I considered that there was no progressive disease.

Subsequently in the following June he had a return of hæmorrhage from lifting a weight (2 cwt.), the blood being ejected on the day following, and to the amount of a pint and a half.

In February 1878, he again applied for advice, and on examining his chest I found the following signs:

Left lung.—Considerable flattening and depression under the clavicle to third rib, with some dulness of percussion, and increased voice sounds with imperfect expansion. Some large moist sounds here, as if the deposit was softening.

The same conditions were found in less degree to the left of the left nipple in the anterior axillary region, and also at the base in the same vertical line. Signs of similar kind, but less marked, were found under the right clavicle, and lower down.

The heart was hypertrophied; apex in usual position, accentuated second sound over the pulmonary artery, but no murmur.

He had a cough now, and was losing flesh.

He was treated with iron and cod-liver oil, and remained under my care until July 1878, when I again examined him.

No moist sound could now be heard at the right apex, and only a slight subcrepitant râle below the right nipple.

Over the left side of the chest were found general harsh and high-pitched crepitations, not increased by cough.

The man had lost 3 lbs. in weight during the last three months, which might in some measure have been due to the very hot weather of the May and June of that year. Otherwise he was in better condition of health, breathing better, and coughing less.

The first stage of hæmorrhage is detected by the presence of fine crepitant râles, due to the insufflation of blood into the alveoli. These râles may continue moist for a considerable period of the time, for weeks, sometimes for two or three months; they gradually lose their liquid character, becoming more viscid and scanty, and at last only a click can be heard after cough.

The blood must then be considered as entering upon the second stage—consolidation, due to clotting. The outlying portions lose their blood by absorption, and the central parts become closely packed by constant pressure.

The physical signs are these:

Dulness of percussion note, increased intensity in the transmitted sounds, with heightened pitch, imperfect expansion, and hesitating delivery of breath sounds; these signs are very local. They are detected in from two months to four months after the hæmorrhage.

Subsequently, considerable retraction of the surface of the chest wall, especially visible in the intercostal spaces, becomes a marked sign.

Finally, the usual signs of softening and excavation are heard during the last stage; the only special characteristics in these cases being the high-pitched sounds, indicative of hard solid tissue in the vicinity.

The physical signs here enumerated not only belong solely to conditions of hæmorrhage, but they remove to another category those signs, dulness of percussion, imperfect expansion, retraction of lung tissue and chest wall, which have often been taken as signs of incipient phthisis; the ground is thus cut away from under the feet of those who ascribe initial or early hæmorrhage to the presence of tubercle in the lungs. Those that are accustomed to investigate the signs of tubercle, and have the opportunity of verifying the diagnosis after death, or of establishing the physical signs during life with the post-

mortem conditions, must be fully aware of the very great difficulty of detecting tubercle, even in large quantities, by the physical signs, still less by any distinct dulness of percussion note.

From a consideration of the physical signs here given, and of the order in which they occur, it is difficult to see what interpretation can be given to them other than that which is derived from pathological evidence.

They are not like those of any ordinary form of phthisis, and they occur, indeed, in exactly a reverse order, for whereas they begin with a fine liquid sound and pass on through various stages of diminished moisture and increased viscidity to the signs of a hard nodular solid occupying the lung in certain localities, the sounds of phthisis are generally supposed to begin from the sounds of hard solid tissue and to pass on to the liquid sounds of breaking down and subsequent excavation.

Those who have watched cases of initial hæmorrhage must be well aware that the physical signs here given are correct, and the hard nodule of the lung may be detected unaltered for years after the occurrence of the hæmorrhage.

To the misinterpretation of the true nature of this hard tissue sound in many cases of hæmorrhage, is due, I believe, the generally received opinion that tubercle is the cause of hæmorrhage ; when it is once recognised that hæmorrhage causes this solid tissue instead of being caused by it, there will be a considerable alteration of opinion as regards the correlation of tubercle and hæmorrhage.

CHAPTER X.

THE CONSEQUENCES OF PULMONARY HÆMORRHAGE.

Almost every systematic writer, says Sir Thomas Watson in his 'Lecture on Pulmonary Hæmorrhage,' quotes, as an example of idiopathic hæmorrhage from the lungs, the story of the Roman governor mentioned by Pliny, who lived to the age of ninety, though he was afflicted with habitual hæmoptysis; and he goes on to say that so hackneyed an example proves the difficulty of finding others.

The real difficulty, however, appears to me to consist in finding individuals of such a position that their condition of health is a matter of common remark, that observations may for that reason extend over a long period of time. Frequent instances of blood-spitting come under notice which cannot be followed up, and if no secondary consequence follows they are not likely to apply again for advice, so that they are entirely lost sight of, unless from their important social position they are the objects of general observation. The dilemma in which I am landed is this: if dependence is placed upon clinical evidence alone it will be urged that pathological proof is required, and it cannot be expected that the pathological department of a hospital devoted to patients suffering from pulmonary disease will afford many examples of patients who have been admitted for, and died from, causes unconnected with this special class of disease.

By an appeal to clinical evidence it is possible to show that blood-spitting occurring without positive evidence of pulmonary phthisis may have either no appreciable effect on the subsequent health of the individual, or its consequences may be so slight as not to constitute a condition of disease, and that this absence of effect may continue for many years.

For the reasons just stated the pathological department of the hospital cannot be laid under contribution for argument in defence of this proposition; but the pathological departments of the general Metropolitan hospitals might furnish a prolific field of observation to any one who would carefully investigate the subject. Let me quote some cases from the 'Clinique Médicale' of Andral:—

Case LXVII.—An old man, aged 71 years, died from cancer of the liver. At the age of 18, 53 years before death, he had spit a great quantity of blood for several months.

No other lesion was found in the lungs than dilatation of some of the bronchial tubes.

Case LXVIII.—A woman, aged 47, died of cancer of the uterus.

For fifteen years before death she scarcely passed three months without spitting a certain quantity of blood.

No appreciable lesion of lungs nor of heart was detected.

Case LXIX.—A young surgeon died of phlebitis after venesection.

Four years before his death he had a tedious pulmonary catarrh, during which he spat blood.

The most careful examination detected no appreciable lesion in the lungs.

Or to take example from a recent work on Phthisis by an author who holds the views of Laennec (Flint on 'Phthisis'):

Case LXX.—Dr. R—— died, at 63 years of age, of cancer of the liver. Thirteen years before he suffered from blood-spitting.

Case LXXI.—Judge C——, 74, died from some cerebral affection.

Had suffered from blood-spitting 9 years before.

To these may be added another series, derived from clinical experience, including instances of individuals attacked with blood-spitting, and examined after a long interval, without evidence of pulmonary disease.

In Ware's cases of hæmoptysis 62 out of 386 were known either to be living in health or had died of diseases not connected with phthisis. His observations extended over the long period of 40 years, and the length of time during which

this immunity from disease had been enjoyed by the several patients varied from 2 to 37 years.

Flint gives evidence of a similar kind: in 61 cases out of 670 the attack of bleeding occurred prior to the commencement of a persistent cough. In 37 cases, the evidence of subsequent phthisis was not established until intervals of varying length had intervened, in one case 16 years, in another 15, in another 10.

The following cases are recorded by Flint as instances of pulmonary bleeding not followed by phthisis:

Mr. B——, age 35. Bleeding profuse and continued for 10 years.

Miss D——, age 22. Bleeding 4 years before visit.

A lady, age 52. Forty attacks of bleeding in 23 years.

A lady, age 35. Profuse bleeding 15 years before.

Mr. W——, age 45. Bleeding 5 years before.

Dr. C——. Bleeding 25 years before. In good health at time of visit.

Mr. W——. Bleeding 28 years before.

Dr. H——. Bleeding 11 years before.

Pollock, in his 'Elements of Prognosis in Consumption,' says: 'I have witnessed the most profuse pulmonary hæmorrhages repeated again and again without any physical signs in the lung of either tubercle, or congestion, without any history of consumption, and without cardiac disease.'

The following cases have been examined by me some time after an attack of copious bleeding from the lungs, and I failed to detect any evidence of phthisis

Name	Age	Time since bleeding	Name	Age	Time since bleeding
Mary B. . . .	40	26 years	William B. . . .	40	14 years
Mary M. . . .	48	10 „	Joseph S. . . .	43	7 „
Jane H. . . .	32	9 „	John S. . . .	29	5 „
Mary A. . . .	38	9 „	Thomas S. . . .	26	5 „
Eliza S. . . .	29	8 „	Alfred S. . . .	25	8 „
Eliza D. . . .	23	6 „	John S. . . .	39	7 „
Jane S. . . .	25	4 „	John B. . . .	29	8 „
Sarah H. . . .	39	4 „	Thomas R. . . .	36	4 „
Eliza D. . . .	14	3 „	Thomas M. . . .	52	4 „
Fanny S. . . .	27	14 „	James C. . . .	39	10 „

The gravity of a case of blood-spitting is proportionate rather

to the amount of pulmonary disease at the time and the stage of phthisis than to the quantity of blood ejected.

Considerable quantities of blood are often raised in initial attacks of bleeding without the patient succumbing to the loss of blood; and I have seen most profuse attacks occur, the patients soon recovering sufficiently to return home.

If the powers of the patient have been much lowered by previous disease the risk is proportionately greater, and if disease of the heart is present the loss of blood in large quantity is serious. But in cases where pulmonary disease is advanced, danger to the patient accrues from the liability to suffocation, arising from want of power to eject the blood, which may be due to a condition of debility arising from the protracted course of enfeebling disease, or to hampered power of coughing when the lung is so invaded by disease, or so intimately adherent to thorax and diaphragm, that the muscles have no opportunity to clear the lungs. In some cases the balance of life is easily upset, and suffocation may ensue from a small hæmorrhage.

Initial bleeding, that is, before the development of phthisis, although it may be profuse, is rarely fatal; but when the stage of destruction is established, then the prognosis in blood-spitting must be always made with great caution.

Even bleedings of a small amount, when there is an old chronic cavity in the lungs, must be regarded with the gravest suspicion, as they may be simply the harbingers of a copious and fatal gush from a ruptured vessel.

The modes of death resulting from loss of blood from the lungs are three: death by fainting, by suffocation from the presence of blood in the bronchial tubes, or by failure of the action of the lungs, no blood being transmitted to the left side of the heart.

The secondary results from an attack of bleeding are the occupation of the lung by blood in more or less quantity, according to the amount of blood raised and the condition of the lung at the time. If the lungs are sound and non-adherent to the chest wall, and the power of coughing is unimpaired, all the blood, if it be not thrown out from a large vessel, may be driven out into the mouth; but if the quantity be profuse, and the bleeding prolonged so as to cause a *besoin de respirer*, the chances

are that some blood is drawn into the lungs and there remains—the fibrinous nodules which are left behind may last for many years without causing any distress or setting up any disease. But if the bleeding lung is very adherent, small and repeated bleedings sometimes result in the occupation of a considerable tract of the lower parts of the lung with blood.

The most usual condition which these blood residues assume is liquefaction, and this occurs with or without the additional presence of a phthisical excavation. In the first case the liquefaction appears to follow admixture with septic matter from the cavity; or it happens as a result of a general tendency, due in all probability to a congestion of the lung tissue in the vicinity, by which the nodules are altered from a solid to a fluid condition.

It is impossible to give any period of time at which such a conclusion is likely to happen, this will depend upon accidental conditions affecting the health of the individual, due to incidental causes, which may occur at any time from exposure to weather, or from any circumstance tending to debilitate.

Pleurisy, with its concomitant pain, may follow the inhalation of blood, especially into the lower axillary portions of the lung, and it is generally in the region below the nipple that local patches of pleurisy may be detected.

From one case which I examined it has appeared to me possible that such a condition of softening might attack these nodules as to perforate their surrounding capsule, and thus, when they are peripheral, produce pneumothorax. The history of this case was unfortunately so incomplete, owing to the state of collapse in which the patient was admitted, that I am unable to quote it as affording positive evidence.

Lastly, it may happen that these nodules, after breaking down, their products having been evacuated, may so leave the vessels which have traversed them, unsupported, that laceration may take place and lead to fatal bleeding. Such a case I examined recently, in which the source of bleeding was traced to a small vessel which ran across a cavity to which were still attached the remains of fibrinous matter, showing the original condition.

If the nodule forcibly separates from the contiguous tissue

by traction, laceration of the surrounding vessels may occur, and I have seen hæmorrhage from this occurrence. It is even possible, from a case which Dr. Goodhart has related to me, that fatal bleeding may take place from this cause.

As regards the secondary formation of tubercle, the amount which I have observed in lungs with cavities formed only from the liquefaction of blood residues has generally been small, and the cases have been few. The amount of tubercle depends, as I believe, in great measure upon the quantity of fluid secretion that is poured out from the walls of a cavity; and as the cavities formed from blood residues do not secrete much, it is not to be expected, if the above rule is valid, that the quantity of tubercle in such cases should be large.

One other consequence must be mentioned, namely, the contraction of the surface of the lung at certain points corresponding to the position of the nodules, with a consequent puckering of the lung surface, and an emphysematous dilatation of the collateral tissue; this is a remarkable consequence of hæmorrhage into the lungs, which has hitherto been regarded without conclusive proof as the result of healed tubercular matter. It may give rise to symptoms and signs of ordinary emphysema during life.

CHAPTER XI.

STATISTICS OF HÆMORRHAGE.

To collect and arrange statistics from a number of cases ready to hand, to calculate percentages, and to strike averages, is a labour at once simple and tedious, than which few are more likely to prove futile and fallacious, unless the work is done with full consideration of the various circumstances and conditions which complicate each case ; and it is with great hesitation that I venture to tread on the track of others who have devoted time and labour to the array of numerical results regarding the relation of phthisis to pulmonary hæmorrhage, and who from their statistics have derived arguments and conclusions which are certainly opposed to my own.

Numerous statistics are already on record, but they have all been drawn up with the preconceived theory that hæmoptysis is the result of the deposit of tubercle supposed in all cases to be the preliminary stage of phthisis.

Nothing is more dangerous than to deal with statistics in this fashion, and inasmuch as I feel that I am quite open to a charge of a similar kind (although in an opposite direction), I shall to some extent make use of statistics compiled by others, in order as far as possible to avoid a proclivity that might unwittingly attach to my arrangement of numbers.

The relative frequency of hæmoptysis in phthisis?—This is a question so often put that it demands an answer, although it is difficult to see how the statistics should be collected. Some cases of phthisis exhibit no signs of hæmorrhage until death is ushered in by a single fatal attack ; others, again, which recover are under observation for a short period, and the ultimate result is not known.

The statistics derived from my cases, as they stand in my note-book, show a very low percentage as compared with those given by others; and the reason for this is partly derived from the fact that numbers of patients attend at the hospital in an early stage of disease, many from long distances, and in subsequent attacks of hæmorrhage they are attended at home, being prevented oftentimes from coming up to the hospital. As I do not attach much value to general statistics of this kind, it will be enough to mention that out of 5,000 cases of well-marked pulmonary disease which would fall naturally under the term 'phthisis,' in 45 per 100 hæmorrhage of a decided kind occurred. In the Appendix will be found the numbers which have been given by others, and some explanation of the differences observed is there offered; but numbers gathered in a somewhat wholesale manner give no satisfactory result, and can lead to no practical conclusion.

It will perhaps be more useful to pick out some of the marked varieties of phthisis, which may be recognised clinically and pathologically, and attempt to solve the question for them.

There are two at least which all authorities agree to recognise as distinct:

1. Acute rapid or galloping phthisis.
2. Scrofulous phthisis.

1. The first kind is now generally considered a tubercular form; it has peculiar clinical and anatomical features, and hæmorrhage is notably absent; a little streaky hæmoptysis may occur during the short course of the disease, but it is not characterised by abundant hæmorrhage. It may also be preceded in some rare instances by copious blood-spitting, but during the course of the disease blood in any quantity is not seen.

Out of 34 cases of this kind which occurred under my observation only 9 were attacked with hæmoptysis.

2. Scrofulous phthisis is that form which occurs in scrofulous patients, it is also characterised by the absence of blood-spitting, except in small quantities, during the course of the disease.

Out of 47 cases of this form of phthisis,

15 were subject to hæmoptysis.

Only those cases of phthisis are here included in which a

distinct history or immediate evidence of glandular disease was obtained. The connection of scrofula with a tendency to hæmorrhage considerably complicates this question, but the scrofulous process is *per se* anti-hæmorrhagic.

3. In addition to those forms of phthisis which are generally stated to be tubercular in character, acute tuberculosis, and those cases of phthisis in which there is an extended invasion of the lung by tubercle, are remarkable for the absence of bleeding during the progress of the tubercle.

4. As regards the syphilitic form of pulmonary disease, which cannot be considered tubercular in character, hæmorrhage is not an infrequent concomitant. I have already, in the 'Lancet' of 1877, given a description (with instances) of that form of pulmonary disease which is found only in syphilitic patients; the symptoms and signs are very peculiar and characteristic, and the cases are not rare in my out-patient practice.

It must be mentioned that in the examination of syphilitic lungs nodules have been assumed to be of specific character because they occurred with a specific history, but to the hæmorrhage to which such patients are prone must be attributed some at least of those nodules which have been considered by many to be gummata.

The relative frequency of hæmoptysis in inherited and non-inherited phthisis.—The difficulty of obtaining accurate information with regard to the inheritance of disease is so great and insuperable that I hesitate to bring forward any additional statistics to those already collected from this point of view. The evidence, as generally obtained, is extremely unsatisfactory, and depends simply upon the memory of the patient, often very defective.

In the cases which form the basis of the following calculations I have purposely omitted all those in which the disease began with hæmoptysis, consequently I have, as far as I could, eliminated chances of error from attributing to the phthisical process what is really due to especial delicacy of structure.

Out of 1,064 cases of well-marked inherited phthisis,

426 suffered from hæmoptysis.

Out of 1,016 cases where phthisis was not known to be inherited,

558 cases had hæmoptysis.

I think it well not to draw any conclusions from these numbers, as there are several considerations which must tend to complicate the question.

The comparative immunity which appears to belong to the members of a phthisical family may be due to the greater care with which such members are invariably watched and tended: the liability to blood-spitting being probably painfully impressed on such individuals from family experience, and hence the necessity of avoiding exertion is fully appreciated. The immunity may be due in part to the fact that inherited phthisis begins at an earlier age, so that the period of exertion is anticipated; or, lastly, it may be due to the tendency to tubercle which such cases freely develope.

Sexual differences.—In addition to these statistics, we have the fact that although trivial hæmorrhages are far more frequent in women than in men, dependent as far as my experience goes on menstrual irregularities, profuse hæmorrhage is far more common in men. In proof of this statement I shall adduce Pollock's statistics:

Of 351 cases of profuse hæmoptysis this author found that 267 were males,

84 were females.

And on this point he says:

'Males are much more subject to spit blood in large quantities than females; on the contrary, in the more moderate degree of hæmoptysis females predominate.'

Now I am not aware of any argument that can be brought forward to support the notion that the processes of phthisis and tubercle are different for the two sexes—I do not say that there are not some features which characterise the two sexes, but I am at present not aware of any distinctions in the process—but I would urge that as the upper lobe of the lungs is more important for the respiratory function in women than in men, that they are less capable of exertion when phthisis is once established, and, moreover, they are far more subject to anæmia, which precludes hæmoptysis. These considerations account in some measure for the difference observed between the sexes, added to which is the liability to the inheritance of weak structure, which from the statistics of hæmophilia appears to belong especially to males.

Sex and age.—These two influences must, as I shall show, be considered together. If they be considered separately, as they have been in the first Medical Report, the only conclusion is, that the female sex is more liable to hæmorrhage than the male in the relation of 65 per cent. to 62 per cent.; but if the tables of sex and age are considered together, it will be seen that there is a very marked liability to hæmorrhage during the early period of a woman's life, while with men the tendency comes later, that is during the period of greatest activity. The subjoined table is taken from the twenty-third table of the first Medical Report:

	Ages							
	0–5	5–15	15–25	25–35	35–45	45–55	55–65	65–75
Males . . .	0	33·3	59·3	71·1	70·6	55·8	23·1	0
Females . .	42·9	78·0	70·4	58·4	58·3	46·7	—	—

This table, which is the result of observation upon 1,084 cases of phthisis, gives the percentage of cases in which hæmoptysis occurred, and it is quite evident that the liability of the two sexes is manifested at different ages. That this is independent of the liability to phthisis is shown by a reference to another table in the report, which shows that the liability to consumption is greatest in both sexes from 25 to 35 years of age, including a term for women below 25, and for men above 35.

Stage of phthisis at which liability to hæmorrhage is greatest.—It appears to me that this point is best shown by collecting a number of cases of both sexes in which the date of hæmoptysis was accurately given and the stage of disease which existed at the time; the subjoined table will show the results obtained. This in great measure supports the argument derived from pathological evidence on the subject that the ulcerative stage is the most liable to bleeding. It is, of course, true that the process continues throughout the formation of cavity, but the answer to such an objection is that exertion is not so easy when much destruction of the lung exists.

The cases are all taken from my own observations, the patients having been carefully examined, and the condition of their lungs noted before the attack of hæmoptysis occurred.

Table showing the stage of phthisis and age at which bleeding occurred in 1,000 *cases.*

Age in years	Men			Total	Women			Total	Grand total
	I.	II.	III.		I.	II.	III.		
1–5	0	1	0	1	0	2	0	2	3
6–10	4	0	0	4	2	2	2	6	10
11–15	1	10	2	13	6	4	7	17	30
16–20	13	30	20	63	20	28	29	77	140
21–25	22	50	32	104	24	46	47	117	221
26–30	17	51	50	118	30	53	30	113	231
31–35	7	38	31	76	18	26	34	78	154
36–40	11	30	20	61	5	15	21	41	102
41–50	7	23	12	42	15	18	12	45	87
51 and over	4	9	5	18	1	1	2	4	22
Total .	86	242	172	500	121	195	184	500	1000
Grand total, stage I. 207—II. 447—III. 256 = 1000.									

The deductions which may be made from the above table are the following:

That the stage of phthisis at which bleeding most frequently occurs is the second stage, that being the period at which the vessels are beginning to break down under the process of ulcerative destruction. This is the period too at which the vessels are least protected, and the greatest amount of lung supplied with vessels remains uninjured.

The above table in no way supports the idea that bleeding is most frequent during the first stage of the phthisical process, while the development of a cavity induces so much difficulty of breathing that exertion is materially impeded, the liability to fatal rupture of vessels which is developed in a few cases from this condition being *pro tanto* diminished.

Influence of temperature.—This cause of hæmoptysis is best shown by statistics for each month in the year. I have taken a thousand cases of well-marked and severe hæmoptysis requiring treatment by styptics; the cases extend over nine years, and the table shows very conclusively that there are special seasons during which hæmoptysis is rife.

The summer months (June and July) are remarkable for the very large proportion of cases; these are presumably due to

summer heat. I may remark that this point was very prominently brought before me this summer, especially during the greatest heat. The cold months of December, January, and February, show the smallest numbers. The increase of numbers in March, April, and November, is to be explained for the first two months probably from the spring winds (east), and for the month of November from the damp fogs which prevail then.

Table showing the occurrence of hæmoptysis arranged according to the time of year.

Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
67	61	90	95	112	130	128	64	64	55	81	63

The main points here brought out by the above table receive confirmation from an examination of the consumption of ergot, which is largely used for hæmorrhage in the hospital. This drug, which has to be used when fresh, has been in use for the last ten years, but only in large quantities since 1874.

In that year 1 lb. of the extract lasted for five months during the first half of the year, whereas in the July and August of the same year 2 lbs. were used.

Again, 1 lb. lasted from September to June 1875, 2 lbs. being again required for July and August.

Two lbs. ordered in September lasted till May 1876, 2 lbs. being used during the June and July of that year.

In the summer quarter of 1877, no less than 6 lbs. were used, and in the May, June, and July of the present year, 4 lbs. were used, whereas 2 lbs. served the subsequent four months.[1]

Gallic acid, which is also largely used for the same purpose, can be kept for a long period, and is ordered in smaller doses, so that the monthly consumption cannot be shown in the same way.

These statistics justify the conclusion that high temperatures encourage hæmoptysis.

On the other hand, cold, at least when not excessive, appears to have no influence in the causation, but tends to repress it. The influence of east winds and of fogs is notorious in their

[1] These numbers are taken from the Hospital Order Book for drugs.

power of increasing bronchial affections, and to these conditions may be due the increased number of cases of hæmoptysis during the months of March, April, and November.

A slight correction should be made if it be thought necessary to calculate the occurrence of hæmoptysis according to the number of patients, and the remarkable decrease in August is to be attributed in great measure to the diminution in the attendances.

The absence of hæmorrhage during the winter months is a very striking point in my experience of nine years; and I believe that, on the whole, a larger number of cases of phthisis do well during the winter than the summer months.

Statistics of pulmonary aneurysm.—It may be of use to add to these statistics some with reference to pulmonary aneurysm, to which fatal bleeding is so often due.

Out of 34 cases collected by Dr. Powell, Dr. Rasmüssen, and myself,

25 occurred among men,

9 among women.

It may occur at any period of life, from three years old up to sixty-four; but the period of life which appears most liable to this disease is between the ages of twenty and thirty.

Out of the 25 cases among males

13 occurred during this period.

Pulmonary aneurysm may give no indications of its presence until the fatal attack, but more frequently it is generally heralded some days, or even weeks, before death by blood-spitting, of which the amount varies, being sometimes scanty, usually profuse. Rupture has been known to end fatally without hæmoptysis.

During the last three years, 383 deaths have occurred in the Hospital, out of which 26 were caused by fatal bleeding. Of these 4 were not examined, and of the remainder, death occurred in one instance from the rupture of an aneurysm into the left bronchus, one from the rupture of the coronary artery of the stomach.

In 5 the source of hæmorrhage was not localised.

In 12 pulmonary aneurysm was the cause of death:

6 of these were in the right lung, and

6 in the left lung.

In 2 cases rupture of an exposed vessel had taken place.

In 1 case the bleeding was due to general pulmonary hæmorrhage.

Of these 22 cases, 20 were males, 2 were females. Mean age 32 years.

The following deductions appear to be a fair result of the statistics here brought forward.

The ultimate causes which lead to pulmonary hæmorrhage are:

1. Exertion, which affects both sexes, but men more than women; not only are instances of this more frequent in men, but the bleeding is more profuse.

2. Heat, which affects both sexes, and induces pulmonary hæmorrhage during the summer months.

3. Alternations of weather, and especially the east winds of spring, and the damp fogs of November, which increase the tendency to bronchial diseases and pulmonary congestions.

4. Functional irregularities of the female sex.

The proximate causes are diseased conditions of the lungs, some of which come under the category of phthisis; in this form of disease, the most destructive stage as regards the effects on the blood-vessels appears to be the second stage, at which period the ulcerative process is most active.

More serious, but less frequent, attacks of bleeding occur towards the end of the disease, when the formation of a cavity has been established.

CHAPTER XII.

THE TREATMENT OF PULMONARY HÆMORRHAGE.

The treatment necessary in any case of bleeding from the lungs must be adapted to the special circumstances of the case; but before the action and uses of various remedies are discussed this fact must be borne in mind, that, in a number of instances, whatever remedy be given, the bleeding is apt to be of short duration: if it results from simple congestion, the unloading of the distended vessels gives relief in many cases; if rupture of a vessel be the cause, and the blood pours from a minute arteriole into the air-cells, the distension of these elastic sacs with blood serves to plug the vessel, and even large cavities are sometimes seen so full of blood that bleeding has been stopped.

In a number of cases then no remedy is necessary, the bleeding is soon over and no harm results; and inasmuch as it is impossible to predict with certainty the amount of blood that will be lost, and whether the bleeding will be of long or short duration, there are probably few points in therapeutics more difficult to determine with satisfaction than the actual and relative value of styptics; hence it frequently happens, whatever remedies are exhibited in the course of an obstinate attack of bleeding, the last remedy tried has the best chance, and the physician who is last called in is likely to have the greatest success: so that something or somebody is praised for labours not their own. This fact is indelibly imprinted on one's mind from the frequency with which patients over and over again detail the satisfactory results of a dose of medicine as a styptic, which was given for some other purpose, and enjoyed no reputation for its astringent properties: indeed, if styptics are used on every occasion when blood makes its appearance in the sputa, a very exalted notion of their value is likely to be

acquired which will suffer a grievous fall when a prolonged and obstinate attack of bleeding is encountered.

The first object of the physician on being called to a case of bleeding is to calm and reassure the patient: a rapid and short examination (only) of the case should be made, and measures will have to be adopted in accordance with the diagnosis; in all cases it is necessary to ascertain the powers of the patient as regards further loss of blood.

Local congestions must be treated according to the condition of the patient and the cause of the disease; if general plethora is present the usual means must be adopted for relieving overloaded vessels: free purging, local and even general blood lettings. Notwithstanding the specious arguments that have been raised against the effects of local applications, they will be found of the greatest service, and I have seen local congestions accompanied by continued bleeding yielding only to the application of a blister. In cases the result of heart disease, in which the mitral valve is inefficient and the heart's action is embarrassed, digitalis is of course our sheet-anchor.

General congestion arising from increased intravascular pressure, whether it result from the plethora of suppressed habitual hæmorrhages or from copious drinking, must be met in the same way; but in cases of the latter kind, advanced cachexia, which sometimes accompanies these profuse bleedings, must be guarded against; and tonics are often useful, none more so than the perchloride of iron. These are cases especially for hygienic treatment, and the diminution of ingesta must be stringently maintained.

The inhalation of turpentine from a piece of lint is of service; and if stimulants are necessary they should be given in the smallest possible bulk.

The copious bleedings which occur from delicacy of structure, the result of inheritance, will not stand any rough treatment, and I do not consider styptics of much avail. Good air, a bracing and rather cold climate, with the frequent administration of ferruginous tonics and cod-liver oil, are far the best treatment for these cases.

The patient should be cautioned against excessive exertion, and temperance in all things should be enjoined.

The actual arrest of bleeding by treatment devised for that special object may be attempted by the following methods:

1. By means adopted for the purpose of diminishing the amount of blood passing into the lungs.

2. By styptics.

1. The method adopted by the ancients, the letting of blood, has to a great extent gone out of favour, probably at a future period it may be restored to that place which it ought to occupy, *nec citra nec ultra*; it should be used in certain cases without hesitation, notably in those plethoric cases in which alarming symptoms of congestion, suffocation, or coma, set in rapidly. If general venesection is not demanded, local blood letting should be employed; and it is evidently better to anticipate impending pulmonary apoplexy than to allow rupture of the vessels and laceration of the pulmonary tissue to take place.

Blood may be prevented from pouring into the right auricle by ligatures applied to the extremities, the most convenient and simple method being the application of tapes to the arms. Flint bears testimony to the value of the method, but it requires attention and care, and the finger should be kept on the patient's pulse to prevent the arrest of blood being carried too far.

Free purging is generally useful, especially in those cases in which the liver is overloaded with blood, and an excessive strain thus thrown on the heart and lungs.

Rest is of the greatest importance, and the patient should be absolutely prevented from all exertion, and from talking; the half-recumbent position should be maintained, and all strain should be avoided, especially with respect to the action of the bowels, for which laxatives are necessary and prohibition of getting out of bed to go to stool. Those who have had the supervision of cases in hospitals must know how often hæmorrhages occur when the patient is straining in this manner.

The administration of food is another point in treatment requiring the attention of the physician. The quantity should be restricted, regular times should be observed for the administration of food, and little should be given at a time; stimulants are better omitted, unless absolutely demanded by the tendency to faintness.

If cod-liver oil has been previously given, it should be intermitted, as it has a marked influence in increasing the fulness of the pulse.

The application of cold is a very important part of the necessary treatment. The temperature of the patient's room should be carefully attended to, and kept at a temperature not above 58° F., and when possible below this.

The application of ice helps greatly to check bleeding, and should be freely applied, care being taken not to chill the patient. It is remarkable how sensitive to heat some patients are who have suffered frequent attacks of bleeding and have become convinced of the remedial advantages of cold. Ice held in the mouth, or the inspiration of air chilled by passing over ice serve the intended object.

It is easy to put on paper recommendations which are known to every practical medical man, but it is difficult enough to get them carried out. The minutiæ of nursing, which cannot be neglected without detriment to the patient, require the constant watchfulness of an intelligent and well trained nurse.

The most satisfactory cases are those which are carefully tended by some friend who has been trained in a proper professional manner, and women who have time at their disposal ought to consider nursing in the light of an accomplishment necessary for their complete education. If this were so, patients would be better nursed, and physicians would have the assurance that their directions were carried out with zeal and intelligence.

2. Treatment by styptics.

With regard to the actual and comparative value of drugs in the arrest of bleeding, I can fully endorse the wise expressions of Flint on this subject, and must say with him, that I find it very difficult to form any positive opinion as to their value severally and relatively: students of medicine are always more confident of success than their teachers, and those who have little experience may be able to give a more decided opinion.

Recently a case came under my care which illustrates the difficulty of the subject:

Case LXXII.—A man with pulmonary phthisis had been sub-

ject to profuse and repeated bleedings from the lungs for several days.

Various remedies had been used by the careful and energetic assistant who had the care of the case. Gallic acid in powder, in saturated aqueous solution, in large quantities—ergot and ergotine, and other drugs in favour as styptics—all without avail, the bleeding continued unabated.

At my suggestion medicines were omitted, food restricted, ice continued as before, and in two days the hæmorrhage abated, and finally stopped.

As a rule powders should not be given perfectly dry, inasmuch as they are very apt to set up cough. The domestic remedy, which is not a bad one, is a spoonful of common salt; powdered alum with the addition of a little water may be given with advantage.

The drugs which are in favour at present are the following:

Gallic acid.	Turpentine.
Mineral acids.	Ergot.

Gallic acid.—On the whole this appears to be as valuable a styptic as any we possess; alone, or, still better, combined with small doses of sulphuric or nitric acids and opium. A very good method of administration is the glycerite, with the addition of a small quantity of spirit to avoid reprecipitation on the addition of water. I cannot say much in favour of pyrogallic acid.

Turpentine is very useful, and may be administered in the usual manner, or by inhalation on a piece of lint.

Ergot.—This drug, which has of late years come into favour as a styptic, has remarkable virtues of a peculiar kind. Sometimes it appears to have a good effect, and there are many reports of its efficacy, but a great drawback to its satisfactory use is the liability to change, which it soon undergoes; so that when the drugs fail of effect, it is uncertain whether the failure is due to faulty preparation, over-keeping, or non-effect. A marked effect in the circulation ought to follow its administration; it should be given in full doses, two drachms of the liquid extract. Of the subcutaneous preparations, that of Morson appears to me a good one; Wood recommends that prepared by Bonjean.

Any good result that may be expected to follow the use of this remedy must be looked for in cases of capillary hæmorrhage, but no drug can be expected to heal an open rupture in a large vessel, or stop a rent sometimes a quarter of an inch long.

Wood, in his work on Therapeutics, speaks with considerable confidence of the effect of ergot as a styptic, and considers that it acts with special rapidity and efficiency in cases of bleeding, when given hypodermically the suddenness of the result being sometimes surprising. But although I have seen instances in which good results have appeared to follow the subcutaneous injection of ergot, I have seen many in which there has been complete failure.

Ipecacuanha has been recommended as of special value, but I believe with Hertz that new bleedings may be produced by the jarring of the body during the act of vomiting, and although it may be given with good effect as long as it only nauseates, not only would the act of vomiting cause a dangerous amount of pressure on the vessels of the lungs, but the faintness which would result from the emetic action of the drug might lead to serious results.

Prevention of hæmoptysis.—It is hardly within the scope of this work to enter fully into details of the climatic treatment of blood-spitting, but there are some broad rules which may be laid down as a guide to a choice of the proper climate.

The cases which require most care are those young persons, generally young men, who are subject to recurrent attacks from structural delicacy of vessels. These demand a cool dry climate, with dry gravelly soil, and heat must be avoided.

The more experience I have in these matters the less am I inclined to expatriate patients who are suffering from pulmonary diseases. There are many things to be considered before advice of such a kind should be given. In many instances it is nothing less than cruel to send away a tender-hearted and affectionate creature from home ties and home comforts: after the novelty of foreign life has worn away, the discomforts which belong to ill-ventilated rooms, rooms without fire-places and with stoves, begin to tell adversely on the patient, and the prolonged absence from home often serves to depress the patient, so that the benefits

of climate have much to counterbalance and sometimes outweigh them.

The same sorts of drawbacks are generally to be found in the sea voyage: the food, the difficulty of getting anything out of hours, the draughty cabins, sometimes do more harm than the sea breezes do good; while many cases which have been watched for years in London could not have done better in any other climate.

Warm climates are certainly contra-indicated for cases of bleeding; in cold climates they do well.

Patients do extremely well at such a place as Hastings during the winter months, and indeed I know no place more advantageous; but during the heat of summer some cooler place must be sought, the mountains of Wales or of Scotland, or the Low Alps of Switzerland. The great object to be obtained is a dry, cool, invigorating air, with such conditions as may induce the patient to be much out of the house, out of stuffy ill-ventilated rooms—walks of sufficient interest to induce exercise, without hills to ascend; and especially the locality should be free to whatever wind may blow, which has much to do with what is called a bracing quality of air. The presence of pine woods is an additional recommendation in the choice of locality.

SUMMARY.

It is perhaps advisable for many reasons to sum up briefly the views which have been advanced in these pages.

Hitherto the occurrence of bleeding from the lungs has been considered to be an indication of the presence of pulmonary phthisis, and by most authors its causation is attributed to the growth of tubercle: to this idea I am strongly opposed.

The consideration of hæmoptysis may be taken from three points of view with reference to the condition of disease in the lungs:

It may be initial, that is, bleeding may occur from the lungs without evidence of previous pulmonary disease, or the disease may be so slight as to be out of all proportion to the bleeding.

In such cases careful investigation of hereditary proclivities will generally show that there is a family tendency to bleeding, which may have been accompanied with phthisis in a previous generation, even if a liability to phthisis be not exhibited in the individual under consideration, and this tendency must be considered as distinct from and not dependent upon the presence of pulmonary phthisis.

Hæmoptysis may be secondary to lung disease, that is, it may be developed in consequence of some destructive process or some morbid condition affecting the balance of circulation in the lungs; it will be then the duty of the physician to consider how far this is accounted for by the amount of pulmonary disease to be discovered at the time, how far it is due to proximate causes, exertion, temperature, alcoholism, and the like, how far it can be considered to be promoted by any hereditary tendency to bleeding that may be elicited from the history of the case.

Hæmoptysis may be also considered as terminal, that is, occurring after the full development of phthisis, as shown by the

presence of excavation. In these cases the occurrence of copious hæmoptysis must always be considered of the gravest import, as it generally occurs from the rupture of a vessel of some size, and from such a condition bleeding often proves fatal.

The two last-named forms of bleeding may and often do occur from the ulcerative action of phthisis; but the presence of tubercle does not cause hæmorrhage in any quantity, nor is it the tubercular process to which copious bleedings can be attributed. The sources of slight and seldom repeated bleedings must always be conjectural, and as they cannot as a rule be considered important as regards the future progress of disease, and may be caused by many different conditions which are very liable to occur during the development of phthisis, it is at least problematical how far and in what cases they can be attributed to tubercular action.

As bleeding may occur from the lungs, and phthisis may be developed afterwards, it is necessary to point out the intermediate steps which connect the two conditions.

During an attack of rapid or repeated bleeding the dyspnœa which is induced by the welling up of the blood in the trachea results in inspiratory gasps often of much force. Under the force of inspiration the blood in the tubes becomes impacted and driven back into various regions of the lungs, and there finally settles as a fibrinous coagulum which loses colour and contracts, and may remain for many years a quite inert body. But under certain secondary conditions affecting the lungs these nodules may soften and share in a general phthisis, or they may be mixed with secretion from a cavity, and so these blood residues may be liquefied and ejected, leaving cavities of a peculiar appearance.

The same thing happens with the calcareous nodules of old hæmorrhages. Hence excavations of the lungs take place, and the matter from these cavities drawn over the bronchial tubes and passed by the inspiratory force into the pulmonary tissue may lead to the development and growth of tubercular nodules.

But in many cases not only does the blood not affect the lungs to such an extent as to leave any residues, but the residues that are left behind may remain unaltered and harmless for many years, and the patients may die from some other disease.

Those who believe that tubercle can produce copious bleedings ought to have some pathological evidence for their belief, and must be able to refer to cases where hæmorrhage has occurred in lungs affected simply with tubercles; and they must explain how it happens that in so many cases of phthisis which are carefully watched, and of which the symptoms are all noted in a book, no indications of bleeding are seen from first to last—why should it happen to these cases thus? it cannot be the process, for it may be a case of scrofulous phthisis or general infection of the lung with tubercle. These difficulties challenge investigation, and some account should be given of them.

If the softening of tubercle be looked to as a cause of bleeding, it is remarkable that the products of this necrosis should be invariably free from the red colouring matter of blood; and when the ulcerative process is distinctly characteristic of tubercle I have seen no proof that extravasations of blood do take place; but I do not pretend to distinguish in all cases between the nontubercular form of ulceration and the continuation of a tubercular form.

I have hitherto seen nothing to convince me that blood *per se* from an individual prone or subject to tubercle can establish tubercle, but my conviction is strong that blood residues which have undergone septic alterations may so contaminate and irritate localities to which the morbid fluid may be transferred as to set up tubercle in those parts, but the amount of tubercle is seldom large.

The views which are here put forward may not meet with favour from those who have already formed their opinions upon this subject on other lines: it may be they will encounter decided opposition; but to those who are entering upon their professional career, or who are pursuing the studies of pathology in large Metropolitan schools where ample opportunities are given for investigating the truth of the opinions here maintained, I would appeal for full and fair consideration. The relations of the forms of disease here discussed are those upon which every medical practitioner is perhaps more often consulted than on any other subject connected with medicine or surgery; and although students are very apt to neglect the study of phthisis, partly owing to the regulations of the Metropolitan

Hospitals, which reject cases of phthisis, and are still more apt to flatter themselves as thoroughly conversant with a complicated disease of which they have seen very few examples, yet they will find that a sound knowledge on the points herein considered is most necessary to them for success in after life; and if they fail to be convinced by the arguments advanced in these pages, let them at least imitate the noble folk of Berea, who received with all readiness of mind and searched daily whether those things were so.

APPENDIX I.

Those that are interested especially in the effect of wind instruments in cases of pulmonary disease, may like to have examples, and the following cases are given as having occurred under my notice:

CASE LXXIII.—Robert B——, 46. Used to play tenor horn, but left off twelve months.
Two years cough. Hæmoptysis a year and a half. Phthisis third stage. Died in two months after visit.

CASE LXXIV.—Robert W——, 25. Clarionet player.
Ill six months. Hæmoptysis. Phthisis second stage.

CASE LXXV.—James K——, 29. Ten years clarionet player.
Two years cough. Never spat blood. Phthisis third stage.

CASE LXXVI.—Frederick C——, 42. Trombone player for twenty-five years.
Nine months cough. No blood. Phthisis second stage.

CASE LXXVII.—Arthur R——, 19. Cornopean player nine months.
Nine months cough. No blood. Phthisis second stage.

CASE LXXVIII.—Harry M——, 37.
Thirteen years in band, and played flageolet, but not for the last three years. Three years cough. Slight hæmoptysis. Phthisis second stage.

CASE LXXIX.—William S——, 47.
Thirteen years trombone player.
Cough two years with hæmoptysis. Phthisis.

CASE LXXX.—William B——, 40.
Cornopean player for five years, from 1854.
Hæmoptysis in 1862. Phthisis second stage.

CASE LXXXI.—Joseph T——, 29. Piccolo player ten years.
No hæmoptysis. Phthisis second stage.

CASE LXXXII.—Charles E——, 37. Clarionet player in band.
No history of bleeding during the time, but two years ago epistaxis, and eighteen months ago hæmoptysis. This was a case of marked inherited tendency to bleeding, which does not appear to have occurred during the time he was in the band.

APPENDIX II.

The following are some of the calculations respecting the comparative frequency of blood-spitting in cases of phthisis derived from different sources:

Walshe represents the frequency at 80·92 per cent. The cases from which these numbers were taken were apparently all in-patients, and consequently this proportion would be high as not including slight cases of phthisis.

Louis found in 87 cases treated in hospital, 57 sufferers from bleeding, giving a percentage of 67.

First medical report of the Brompton Hospital, from a calculation of the in-patients, gives 63.

Second medical report gives 62.

Dr. Pollock, in his 'Elements of Prognosis,' gives 58·4 out of 1,200 out-patient cases.

Dr. C. T. Williams, out of 1,000 private cases, found 569 affected with bleeding, a percentage of 57.

Dr. Cotton, in his 1,000 cases derived from the in-patients of the hospital, gives 53.

The calculations derived solely from the consideration of a number of in-patients, who are admitted for the pulmonary disease in an advanced stage, are manifestly derived too much from advanced cases, and the percentage will be high.

The numbers, derived as my own are from out-patients solely, may possibly be too low, but I confess I see no way of arriving at any common ground, and many cases among our out-patients are in the last stage of phthisis.

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